

This electronic thesis or dissertation has been downloaded from the King's Research Portal at <https://kclpure.kcl.ac.uk/portal/>



Investigating the efficacy of a self-management intervention for people with chronic rheumatoid arthritis : using an illness representations approach to understand change processes and outcome

Pimm, Theo John

The copyright of this thesis rests with the author and no quotation from it or information derived from it may be published without proper acknowledgement.

END USER LICENCE AGREEMENT



Unless another licence is stated on the immediately following page this work is licensed

under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International

licence. <https://creativecommons.org/licenses/by-nc-nd/4.0/>

You are free to copy, distribute and transmit the work

Under the following conditions:

- Attribution: You must attribute the work in the manner specified by the author (but not in any way that suggests that they endorse you or your use of the work).
- Non Commercial: You may not use this work for commercial purposes.
- No Derivative Works - You may not alter, transform, or build upon this work.

Any of these conditions can be waived if you receive permission from the author. Your fair dealings and other rights are in no way affected by the above.

Take down policy

If you believe that this document breaches copyright please contact librarypure@kcl.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.

**INVESTIGATING THE EFFICACY OF
A SELF-MANAGEMENT
INTERVENTION FOR PEOPLE WITH
CHRONIC RHEUMATOID
ARTHRITIS: USING AN ILLNESS
REPRESENTATIONS APPROACH TO
UNDERSTAND CHANGE PROCESSES
AND OUTCOME**

Theo John Pimm

A Dissertation submitted in fulfillment of the degree of Doctor in Philosophy, King's
College, University of London.

June, 2003.



Acknowledgements

I would like to thank the following people for their help in completing this thesis. The participants who gave so much time and provided us with so many ideas. My clinical and research colleagues at the Oxford Regional Rheumatic Diseases Research Centre, Stoke Mandeville Hospital who helped in the recruitment of subjects and assisted with the running of the Arthritis Self Management programmes, especially Mary Amos, Jackie Hall, Jan Hann, Robert Field, Katie Glover and in particular, Dr. Margaret Byron. I would like to thank the Assistant Psychologists who have helped me with collection of data and preparation of this manuscript, especially Deborah Curson, Sharon Hill, Pam Jones, Rina Ediwerera, Jay Lipmann, Genevieve Aikman, Catherine Bamford and Nicky Jackson. I would like to thank my clinical colleagues who have supported me and covered my clinical commitments during the final weeks of writing, especially Bronwyn Carter and Imogen Cotter. A special thanks to my secretary, Linda Rogers, for her help in preparation of the manuscript. I am especially grateful for the help of Ann pepper, my Personal Reader, who has read so many articles and books on to tape for me. I would also like to thank Bill Mitchell who proofread the manuscript.

I would like to thank the Arthritis Research Campaign (ARC) for their financial support for this project (grant numbers PO503 and PO520) and also for providing me with grants to enable me to present the work at International meetings. I would like to thank the Royal Society of Medicine for awarding me the Kovacs Travelling Fellowship that enabled me to visit research centres conducting similar research in the USA. I would also like to thank the researchers that I visited, for their help, especially Professor Matthew Liang, Dr. Lauren Daltroy, Professor Brenda DeVellis, Dr. Robert DeVellis, Professor Frank Keef, Professor Kate Lorig and Professor Howard Leventhal.

I am grateful to Professor Kate Lorig, who was kind enough to allow me to use and modify the course materials for the Arthritis Self-Management programme.

I would like to thank Matthew Hankins for statistical advice and Professor John Weinman who has been a stimulating, patient and supportive Supervisor.

Finally, and most importantly, I would like to thank my wife Janice and my daughter Aisha for their tolerance while I have been writing this dissertation.

TABLE OF CONTENTS

1: The Challenge of Rheumatoid Arthritis... .. 1

1.1 Introduction... .. 1

1.2 Clinical features of RA... .. 1

1.3 Epidemiology... .. 2

1.3.1 Epidemiology of Arthritis... .. 2

1.3.2 Incidence of RA... .. 2

1.3.3 Prevalence of RA... .. 3

1.3.4 Epidemiological trends... .. 4

1.4 Pathogenesis of RA... .. 4

1.5 Course and prognosis of RA... .. 6

1.6 Health outcomes in RA... .. 10

1.6.1 Mortality... .. 11

1.6.2 Symptomatology... .. 12

1.6.2(a) Pain... .. 12

1.6.2(b) Fatigue... .. 14

1.6.2(c) Sleep... .. 15

1.6.2(d) Joint stiffness... .. 16

1.6.3 Physical disability... .. 16

1.6.4	<i>Emotional distress...</i>	20
1.6.5	<i>Work disability...</i>	25
1.7	The cost of RA...	27
1.8	Treatment and management of RA...	28
1.9	Summary...	31
2:	Psychological Processes in the Course and Outcome of Rheumatoid Arthritis...	32
2.1	Introduction...	32
2.2	The biopsychosocial model...	33
2.3	Leventhal's self-regulatory model...	35
2.4	Application of the self-regulatory framework to RA...	38
2.5	Psychosocial processes in adaptation to rheumatic disease...	39
2.5.1	<i>Illness related cognitions and appraisals...</i>	39
2.5.2	<i>Coping procedures...</i>	43
2.5.3	<i>Appraisals of coping efficacy...</i>	48
2.5.3(a)	<i>Catastrophising...</i>	49
2.5.3(b)	<i>Self-efficacy...</i>	51
2.5.3(c)	<i>Helplessness...</i>	53
2.5.4	<i>Social context...</i>	56
2.5.4(a)	<i>Social support...</i>	56
2.5.4(b)	<i>Interpersonal and marital relationships</i>	60
2.5.4(c)	<i>Social comparisons...</i>	61
2.5.4(d)	<i>Socio-economic status...</i>	62

2.6	Review of research using the self-regulatory model in RA...	63
2.6.1	<i>The development of illness representations...</i>	63
2.6.2	<i>Self-regulatory processes in chronic RA...</i>	65
2.7	Summary...	70
3:	Psycho-educational interventions for people with RA...	72
3.1	Introduction...	72
3.2	Rationale for psycho-educational interventions...	72
3.3	Definition and aims of psycho-educational intervention...	75
3.4	Psycho-educational intervention methods...	76
3.5	Efficacy of psycho-educational interventions...	80
3.5.1	<i>Educational interventions...</i>	80
3.5.2	<i>Self-management interventions...</i>	81
3.5.3	<i>Cognitive behavioural interventions...</i>	84
3.5.4	<i>Other psychosocial interventions...</i>	89
3.5.5	<i>Reviews and meta-analyses of psycho-educational interventions...</i>	89
3.6	Mechanisms of change in psycho-educational interventions...	100
3.7	Summary and future research...	105
4.	Summary of the literature and implications for the present study...	107
4.1	Clinical context for the study...	107
4.2	The efficacy of self-management intervention for people with chronic RA...	107

4.3	The relationship between illness representations coping and health outcomes in chronic RA... ..	109
4.4	The role of illness representations and coping in moderating or mediating changes in self-management intervention... ..	110
4.5.	Research questions... ..	111
4.6	Aims and hypotheses... ..	111
4.6.1	<i>The efficacy of self-management intervention for people with chronic RA... ..</i>	111
4.6.2	<i>The relationship between illness representations coping and health outcomes in chronic RA... ..</i>	112
4.6.3	<i>The role of illness representations and coping in moderating and mediating outcomes of a self-management intervention... ..</i>	114
5:	Method... ..	115
5.1	Design... ..	115
5.2	Procedure... ..	115
5.3	Participants... ..	117
5.4	Assessment process... ..	118
5.5	Measures... ..	119
5.5.1	<i>Selection of outcome measures... ..</i>	119
5.5.2	<i>Demographic data... ..</i>	121

5.5.3	<i>Primary outcome measures...</i>	121
5.5.3(a)	<i>Pain.....</i>	121
5.5.3(b)	<i>Physical disability.....</i>	122
5.5.3(c)	<i>Emotional state.....</i>	123
5.5.4	<i>Secondary outcomes.....</i>	125
5.5.4(a)	<i>Social and occupational function.....</i>	125
5.5.4(b)	<i>Individualized quality of life (PGI).....</i>	125
5.5.4 (c)	<i>Disease activity.....</i>	127
5.5.4(d)	<i>Health care utilization.....</i>	128
5.5.5	<i>Self-regulatory variables.....</i>	128
5.5.5(a)	<i>Illness representations.....</i>	128
5.5.5(b)	<i>Self-efficacy.....</i>	130
5.5.5(c)	<i>Coping procedures.....</i>	131
5.6	<i>Detailed procedure.....</i>	133
5.7	<i>The Self-management intervention.....</i>	136
5.8	<i>Ethical considerations.....</i>	137
5.8.1	<i>Confidentiality.....</i>	137
5.8.2	<i>Informed consent.....</i>	138
5.8.3	<i>Adverse effects.....</i>	138
5.8.4	<i>Withholding intervention.....</i>	139
5.8.5	<i>Debriefing.....</i>	139
6:	<i>Results: Overview, sample characteristics and representativeness</i>	140
6.1	<i>Overview of results.....</i>	140

6.2	Statistical methods - sample characteristics and representativeness.....	141
6.3	Eligibility.....	142
6.4	Response rates and comparison of participants and non-participants.....	144
6.5	Comparison of participants who provided data with those who provided no data.....	147
6.6	Withdrawal rates.....	150
6.7	Comparison of participants who completed the study and those who provided no data or withdrew.....	152
6.8	Comparison of participants who completed the study and those who withdrew during the study.....	155
6.9	Missing data.....	159
6.10	Comparison of participants in the treatment group who completed treatment and those that did not.....	159
6.11	Demographic characteristics of the total sample and comparison of demographic characteristics of treatment and control groups.....	164
6.12	Research Question 1.1.....	167
6.13	Research Question 1.2.....	172
6.14	Research Question 1.3.....	175
6.15	Comparison of treatment and control groups at baseline on main response variables.....	176
6.16	Sample characteristics and representativeness: summary of findings.....	178
7:	The efficacy of a self-management intervention for people with chronic RA.....	182
7.1	Statistical methods.....	182

7.2	Changes in health outcomes following intervention.....	183
	7.2(a) <i>Pain</i>	184
	7.2(b) <i>Physical disability</i>	188
	7.2(c) <i>Emotional distress</i>	191
	7.2(d) <i>Social and occupational function</i>	194
	7.2(e) <i>Individualized quality of life (PGL)</i>	197
	7.2(f) <i>Disease activity</i>	198
	7.2(g) <i>Health care utilization</i>	205
7.3	Changes in illness representations and coping procedures following self-management intervention.....	206
	7.3(a) <i>Illness identity</i>	206
	7.3(b) <i>Symptom coherence</i>	207
	7.3(c) <i>Time line</i>	208
	7.3(d) <i>Consequences</i>	209
	7.3(e) <i>Cause</i>	210
	7.3(f) <i>Control/cure</i>	211
	7.3(g) <i>Self-efficacy</i>	212
	7.3(h) <i>Coping strategies</i>	215
	7.3(i) <i>Self management behaviours</i>	225
7.4	Summary of findings.....	234
	7.4.1 <i>Research Question 1.4 And Hypothesis 1.1</i> <i>Summary Of findings</i>	234
	7.4.2 <i>Research Question 1.5 and Hypothesis 1.2</i> <i>Summary of findings</i>	236

8:	The relationship between illness representations, coping and health outcomes.....	241
8.1	Introduction.....	241
8.2	Statistical methods.....	241
8.3	The relationship between illness representations and coping procedures.....	244
8.4	The relationship between illness representations and coping procedures, and health outcomes at baseline.....	247
8.4.1	<i>Explaining baseline pain.....</i>	248
8.4.2	<i>Explaining baseline pain (excluding IPQ Identity and IPQ Coherence).....</i>	251
8.4.3	<i>Explaining baseline physical disability.....</i>	254
8.4.4	<i>Explaining baseline emotional distress.....</i>	256
8.4.5	<i>Explaining baseline social/occupational function... ..</i>	259
8.4.6	<i>Explaining baseline individualized quality of life... ..</i>	261
8.4.7	<i>Explaining baseline disease activity.....</i>	263
8.4.8	<i>Explaining baseline health care utilization.....</i>	267
8.4.9	<i>Hypothesis 2.2 summary of findings.....</i>	267
8.5	Do baseline illness representations and coping procedures predict future health outcomes?.....	270
8.5.1	<i>Predicting pain time 3, 4 and 5.....</i>	271
8.5.2	<i>Predicting pain time 3, 4 and 5 (excluding IPQ Identity and IPQ Coherence).....</i>	274
8.5.3	<i>Predicting physical disability time 3, 4 and 5.....</i>	276
8.5.4	<i>Predicting emotional distress time 3, 4 and 5.....</i>	277

8.5.5	<i>Predicting social and occupational function time 3, 4 and 5.....</i>	279
8.5.6	<i>Predicting individualized quality of life time 3, 4 and 5.....</i>	279
8.5.7	<i>Predicting disease activity time 3, 4 and 5</i>	280
8.5.8	<i>Predicting health-care utilization time 3, 4 and 5 ...</i>	282
8.5.9	<i>Hypothesis 2.3 summary of findings.....</i>	283
8.6	<i>Do changes in illness representations and coping procedures predict future health outcomes?.....</i>	289
8.6.1	<i>Predicting pain time 3, 4 and 5.....</i>	290
8.6.2	<i>Predicting pain time 3, 4 and 5 (excluding IPQ Identity and IPQ Coherence).....</i>	293
8.6.3	<i>Predicting physical disability time 3, 4 and 5.....</i>	295
8.6.4	<i>Predicting emotional distress time 3, 4 and 5.....</i>	296
8.6.5	<i>Predicting social/occupational function time 3, 4 and 5.....</i>	298
8.6.6	<i>Predicting individualized quality of life time 3, 4 and 5.....</i>	299
8.6.7	<i>Predicting disease activity time 3, 4 and 5.....</i>	300
8.6.8	<i>Hypothesis 2.4 summary of findings.....</i>	301
9:	<i>The role of illness representations and coping in mediating/moderating changes in a self-management intervention... ..</i>	306
9.1	<i>Introduction... ..</i>	306
9.2	<i>Statistical methods.....</i>	307

9.3	Do illness representations and coping procedures moderate the effect of a self-management intervention?.....	310
9.3.1	<i>Individualized Quality Of Life</i>	310
9.3.2	<i>Self-efficacy for other arthritis symptoms</i>	311
9.4	Do illness representations and coping procedures mediate the effect of a self-management intervention?.....	314
9.4.1	<i>Individualized quality of life</i>	315
9.4.2	<i>Self-efficacy for pain</i>	316
9.4.3	<i>Self-efficacy for other arthritis symptoms</i>	317
9.5	Hypotheses 3.1 and 3.2 Summary of findings.....	318
9.5.1	<i>Hypothesis 3.1: Summary of findings</i>	318
9.5.2	<i>Hypothesis 3.2: Summary of findings</i>	319
10:	Discussion.....	320
10.1	Introduction.....	320
10.2	The efficacy of a self-management intervention for people with chronic RA.....	321
10.3	The relationship between illness representations, coping and health outcomes in chronic RA.....	330
10.3.1	<i>Relationships between illness representations and coping</i>	330
10.3.2	<i>Do illness representations and coping explain health outcomes at baseline?</i>	332
10.3.3	<i>Do illness representations and coping predict future health outcomes?</i>	336

10.3.4	<i>Do changes in illness representations and coping predict health outcomes?</i>	339
10.4	Do illness representations or coping procedures moderate or mediate the effects of a self-management intervention.....	344
10.5.	Methodological considerations and study limitations.....	349
10.5.1	<i>Design.....</i>	349
10.5.2	<i>Sample size.....</i>	350
10.5.3	<i>Lack of participant blinding and attention placebo control group.....</i>	352
10.5.4	<i>Failure of randomisation.....</i>	354
10.5.5	<i>Floor effects.....</i>	355
10.5.6	<i>Assessment of change in individualized quality of life</i>	356
10.5.7	<i>Measurement of illness representations.....</i>	356
10.5.8	<i>Multiple comparisons.....</i>	359
10.5.9	<i>Direction of causation.....</i>	359
10.5.10	<i>Comprehensive self-management intervention.....</i>	360
10.5.11	<i>Maintenance of treatment gains.....</i>	361
10.5.12	<i>Generalisation of findings.....</i>	361
10.5.13	<i>Possible adverse treatment effects.....</i>	363
10.5.14	<i>Ecological validity and cost-effectiveness.....</i>	364
10.6	Theoretical implications.....	365
10.6.1	<i>Self-efficacy and Social Learning Theory.....</i>	365
10.6.2	<i>Generic self-regulatory models.....</i>	370
10.6.3	<i>Implications of the findings for the SRM.....</i>	370

10.7 Future research and treatment implications..... 376

10.7.1 Who is likely to benefit from intervention? 376

10.7.2 When is intervention likely to be effective? 377

10.7.3 What mechanisms facilitate successful adaptation and
response to treatment 378

10.7.4 Which components of treatment are necessary? 380

10.7.5 How should interventions be delivered? 381

10.7.6 Do interventions have other benefits or adverse
effects? 382

10.7.7 Leventhal's SRM..... 383

10.8 Summary of findings and conclusions..... 384

11: References..... 387

LIST OF TABLES

	Page
Chapter 3:	
Table 3.1. Pooled effect sizes for six outcomes from a meta-analysis of psychological interventions for RA.... ..	96
Chapter 6:	
Table 6.1. Number of ineligible participants in the sample for each inclusion and exclusion criterion.....	142
Table 6.2. Comparison of sex, age, duration of illness and distance from research centre for study participants and people who did not participate.....	146
Table 6.3. Reasons given for not participating in the study.....	147
Table 6.4. Comparison of sex, age, duration of illness, distance from research centre and experimental condition for participants who provided no data and those that provided date.....	149
Table 6.5. Reasons for providing no data given by the 18 participants who provided no data.....	150
Table 6.6. Frequency and cumulative frequency of withdrawals from the study at each assessment point for the treatment group, control group and total sample.....	151
Table 6.7. Comparison of sex, age, duration of illness, distance from research centre, and experimental condition for participants who provided no data or withdrew and those that completed the study.....	153
Table 6.8. Comparison of sex, age, duration of illness, distance from research centre and experimental condition for participants who withdrew and those that completed the study.....	154
Table 6.9. Results of T-tests showing significant differences between participants who withdrew and those that completed the study.	156

Table 6.10	Reasons for withdrawal given by the 13 participants who withdrew during the study.....	157
Table 6.11.	Frequency of missing data at each assessment point for the treatment group, control group and total sample.....	158
Table 6.12.	Treatment attendance categorised to show frequency of participants not starting, not completing and completing treatment.....	160
Table 6.13.	Comparison of number of treatment sessions attended and the frequency of participants withdrawing from the study at each time point.....	160
Table 6.14.	Comparison of participants in the Treatment Group who attended 0-3 treatment sessions and those attending 4-6 sessions with respect to sex, age, duration of illness and distance from research centre.....	162
Table 6.15.	Significant T-test Results comparing participants in treatment group who attended 0-3 sessions with those who attended 4-6 sessions, on baseline means of main response variables.....	163
Table 6.16.	Frequency and percentage of participants Attending each group Treatment session.....	163
Table 6.17.	Demographic characteristics of the sample (sex, marital status, employment and ethnic origin) and results of statistical analyses testing for differences between treatment and control groups.	165
Table 6.18.	Demographic characteristics of the sample (age, years of education, duration of illness and number of children and results of statistical analyses testing for differences between treatment and control groups.....	166
Table 6.19.	Details of participants' marital status.....	166
Table 6.20.	Details of participants' employment status.....	167
Table 6.21.	Frequency and percentage of participants within and outside of normal ranges for blood tests.....	168
Table 6.22.	Frequency distribution for HAQ scores.....	169

Table 6.23.	HADS Anxiety Baseline frequency and percentage of participants below and above cut off for possible and definite anxiety by experimental group and gender.....	170
Table 6.24.	HADS Depression Baseline frequency and percentage of participants below and above cut offs for possible and definite depression by experimental group and gender.....	171
Table 6.25.	Significant T-test results comparing control and treatment groups on baseline means of main response variables.....	177
Table 6.26.	Significant Mann-Whitney U results comparing control and treatment groups on baseline means of main response variables.	177
Table 6.27.	Summary of results of statistical analyses comparing study participants and those who declined to participate, provided no data, withdrew or did not complete treatment and treatment and control groups.....	179
Chapter 8:		
Table 8.1.	Correlations of time 1 IPQ and cope variables.....	246
Table 8.2.	Summary of cross sectional multiple regression analyses explaining pain at time 1 (IPQ Identity and Coherence included in analyses).....	250
Table 8.3.	Summary of cross sectional multiple regression analyses explaining pain at time 1 (excluding IPQ Identity and IPQ Coherence).....	253
Table 8.4.	Summary of cross sectional multiple regression analyses explaining disability at time 1.....	255
Table 8.5.	Summary of cross sectional multiple regression analyses explaining emotional distress at Time 1.....	258
Table 8.6.	Summary of cross sectional multiple regression analyses explaining social and occupational function at Time 1.....	260
Table 8.7.	Summary of cross sectional multiple regression analyses explaining individualized quality of life at Time 1.....	262

Table 8.8.	Summary of cross sectional multiple regression analyses explaining disease activity at Time 1.....	266
Chapter 9:		
Table 9.1	Summary of multiple regression analyses investigating moderator effects.....	313
Table 9.2.	Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on individualized quality of life at time 3.....	315
Table 9.3.	Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on self-efficacy for pain at time 4.	316
Table 9.4.	Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on self-efficacy for other arthritis symptoms at time 3.....	317
Appendix I:		
Table I.1	Baseline scores on measures of pain for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.	
Table I.2	Baseline scores on measures of physical disability for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.	
Table I.3	Baseline scores on measures of emotional distress for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.	
Table I.4	Baseline scores on measures of social and occupational function for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.	

Table I.5	Baseline scores for individualized quality of life for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Table I.6	Baseline scores on measures of disease activity for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Table I.7	Baseline scores on measures of health care utilization for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Table I.8	Baseline means and standard deviations for illness perceptions questionnaire for total sample, treatment and control groups, alpha reliability coefficients and results of t-tests comparing treatment and control groups.
Table I.9	Baseline scores on measures of self-efficacy for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Table I.10	Baseline scores on measures of coping for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Table I.11	Baseline scores on measures of self-management behaviours for total sample, treatment and control groups and results of t-tests comparing treatment and control groups.
Appendix J:	
Table J.1:	Results of ANOVAs and ANCOVAs testing hypothesis 1.1a-g.
Table J.2:	Hypotheses 1.1a-g Within participants main effects and interaction effects from planned contrasts comparing time 1 vs 2, 2 vs 3, 3 vs 4, and 4 vs 5.
Table J.3:	Hypotheses 1.1a-g within participants main effects and interaction effects from planned contrasts comparing time 1 vs 3, 1 vs 4 and 1 vs 5.
Table J.4:	Results of ANOVAs and ANCOVAs testing hypothesis 1.2 a-i.

Table J.5	Hypotheses 1.2a-i within participants main effects and interaction effects from planned contrasts comparing time 1 vs 2, 2 vs 3, 3 vs 4, and 4 vs 5.
Table J.6	Hypotheses 1.2a-i within participants main effects and interaction effects from planned contrasts comparing time 1 vs 3, 1 vs 4 and 1 vs 5.

Appendix K:

Table K.1.	Summary of multiple regression analyses predicting pain at time 3, 4 and 5, IPQ Identity and IPQ Coherence included in analyses.
Table K.2.	Summary of multiple regression analyses predicting pain at time 3, 4 and 5. (excluding IPQ Identity and IPQ Coherence).
Table K.3.	Summary of multiple regression analyses predicting disability at time 3, 4 and 5.
Table K.4.	Summary of multiple regression analyses predicting emotional distress at time 3, 4 and 5.
Table K.5.	Summary of multiple regression analyses predicting social and occupational function at time 3, 4 and 5.
Table K.6.	Summary of multiple regression analyses predicting individualized quality of life at time 3, 4 and 5.
Table K.7.	Summary of multiple regression analyses predicting disease activity at time 3, 4 and 5.
Table K.8.	Summary of multiple regression analyses predicting health care utilization at time 3, 4 and 5.

LIST OF FIGURES

	Page
Figure 2.1. The biopsychosocial model of arthritis pain and disability.....	34
Figure 2.2. Leventhal’s Self-Regulatory Model (SRM).....	37
Figure 5.1. Zelen’s modified design for randomised clinical trials.....	116
Figure 6.1. Graph showing control, treatment & total sample mean & 95% confidence intervals for symptoms reported on IPQ Identity at time 1.....	173
Figure 6.2. Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ scales at time 1.....	173
Figure 6.3. Graph showing frequency of control, treatment & total sample participants reporting different numbers of causal attributions on the IPQ at time 1.....	174
Figure 6.4. Graph showing frequency of control, treatment & total sample participants reporting different types of causes at time 1.....	174
Figure 6.5. Graph showing control, treatment & total sample mean & 95% confidence intervals for Cope sub scales at time 1.....	175
Figure 7.1. Graph showing control, treatment & total sample mean & 95% confidence intervals for NRIP scores between time 1 & 5	186
Figure 7.2. Graph showing control, treatment & total sample mean & 95% confidence intervals for GRSP scores between time 1 & 5	186
Figure 7.3. Graph showing control, treatment & total sample mean & 95% confidence intervals for BVAS scores between time 1 & 5	187
Figure 7.4. Graph showing control, treatment & total sample mean & 95% confidence intervals for pain distress scores between time 1 & 5.....	187

Figure 7.5. Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Symptom scores between time 1 & 5..... 188

Figure 7.6. Graph showing control, treatment & total sample mean & 95% confidence intervals for HAQ scores between time 1 & 5 190

Figure 7.7. Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Physical scores between time 1 & 5..... 191

Figure 7.8. Graph showing control, treatment & total sample mean & 95% confidence intervals for Hospital Anxiety scores between time 1 & 5..... 193

Figure 7.9. Graph showing control, treatment & total sample mean & 95% confidence intervals for Hospital Depression scores between time 1 & 5..... 193

Figure 7.10. Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Affect scores between time 1 & 5..... 194

Figure 7.11. Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Role scores between time 1 & 5..... 196

Figure 7.12. Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Social scores between time 1 & 5..... 196

Figure 7.13. Graph showing control, treatment & total sample mean & 95% confidence intervals for Patient Generated Index scores between time 1 & 5..... 198

Figure 7.14. Graph showing control, treatment & total sample mean & 95% confidence intervals for Early Morning Stiffness scores between time 1 and 5..... 201

Figure 7.15. Graph showing control, treatment & total sample mean & 95% confidence intervals for CRP scores between time 1 & 5 201

Figure 7.16.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Tender Joint Score (Ritchie Articular Index) between time 1 & 5.....	202
Figure 7.17.	Graph showing control, treatment & total sample mean & 95% confidence intervals for WCC scores between time 1 & 5.	202
Figure 7.18.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Platelet scores between time 1 & 5	203
Figure 7.19.	Graph showing control, treatment & total sample mean & 95% confidence intervals for HB scores between time 1 & 5....	203
Figure 7.20.	Graph showing control, treatment & total sample mean & 95% confidence intervals for ESR scores between time 1 & 5....	204
Figure 7.21.	Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of Dr Visits between time 1 & 5....	206
Figure 7.22.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Identity scores between time 1 & 5	207
Figure 7.23.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Coherence scores between time 1 & 5.....	208
Figure 7.24.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Time line between time 1 & 5.....	209
Figure 7.25.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Consequences scores between time 1 & 5.....	210
Figure 7.26.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Cause scores between time 1 & 5	211
Figure 7.27.	Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Control/Cure scores between time 1 & 5.....	212
Figure 7.28.	Graph showing control, treatment & total sample mean & 95% confidence intervals for self-efficacy for pain scores between time 1 & 5.....	213

Figure 7.29.	Graph showing control, treatment & total sample mean & 95% confidence interval for self-efficacy for function scores between time 1 & 5.....	214
Figure 7.30.	Graph showing control, treatment & total sample mean & 95% confidence intervals for self-efficacy for other arthritis symptoms scores between time 1 & 5.....	214
Figure 7.31.	Graph showing control, treatment & total sample mean & 95% confidence interval for COPE:positive interpretation/growth scores between time 1 & 5.....	218
Figure 7.32.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE:Active coping scores between time 1 & 5.....	219
Figure 7.33.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Planning scores between time 1 & 5.....	219
Figure 7.34.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Seeking emotional social support scores between time 1 & 5.....	220
Figure 7.35	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Seeking instrumental social support between time 1 & 5.....	220
Figure 7.36.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Suppression of competing activities scores between time 1 & 5.....	221
Figure 7.37.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Acceptance scores between time 1 & 5.....	221
Figure 7.38.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Mental disengagement scores between time 1 & 5.....	222
Figure 7.39.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Focus/vent emotions scores between time 1 & 5.....	222

Figure 7.40.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Behavioural disengagement scores between time 1 & 5	223
Figure 7.41.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Denial scores between time 1 & 5.....	223
Figure 7.42.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Restraint coping between time 1 & 5.....	224
Figure 7.43.	Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Humour scores between time 1 & 5.....	224
Figure 7.44.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Stretching exercises scores between time 1 & 5.....	228
Figure 7.45.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Strengthening exercises scores between time 1 & 5.....	228
Figure 7.46.	Graph showing control, treatment & total sample mean & 95% confidence interval for Relaxation scores between time 1 & 5.....	229
Figure 7.47.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Massage scores between time 1 & 5.....	229
Figure 7.48.	Graph showing control, treatment & total sample mean & 95% confidence intervals for frequency of walks between time 1 & 5.....	230
Figure 7.49.	Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins walked between time 1 & 5.....	230
Figure 7.50.	Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of miles walked between time 1 & 5.....	231

Figure 7.51.	Graph showing control, treatment & total sample mean & 95% confidence intervals for Frequency of cycling between time 1 & 5.....	231
Figure 7.52.	Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins cycled between time 1 & 5	232
Figure 7.53.	Graph showing control, treatment & total sample mean & 95% confidence intervals for frequency of swimming scores between time 1 & 5.....	232
Figure 7.54	Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins swimming scores between time 1 & 5.....	233

List of Appendices

Appendix A	The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis
Appendix B	Confirmation of ethical approval
Appendix C	Grant proposal
Appendix D	Inclusion /Exclusion criteria
Appendix E	Patient information sheet, consent forms, GP letters
Appendix F	Questionnaires
Appendix G	Debrief
Appendix H	Treatment manual
Appendix I	Baseline descriptive statistics and T-tests for main response variables
Appendix J	Results of ANOVAs and ANCOVAs testing Hypotheses 1.1 and 1.2
Appendix K	Results of Multiple Regression Analyses testing Hypotheses 2.3 and 2.4

Abstract

Rheumatoid arthritis (RA) frequently leads to chronic distressing symptoms and severe disabilities despite medical treatment. Psycho-educational interventions for people with RA have been shown to increase understanding of RA, adaptive cognitions and coping behaviours and, less consistently, to improve health outcomes. There is evidence that the health outcome benefits of psycho-educational interventions may be mediated by change in psychological variables.

The study aimed to investigate:

Does an UK RA self-management intervention produce therapeutic benefits for hospital outpatients with chronic RA?

Is Leventhal's self-regulatory model useful for understanding processes by which people perceive and respond to RA?

Are the benefits of a self-management intervention explained by illness representations or coping procedures?

Participants, in this single-blind randomised controlled trial, were 136 hospital outpatients with RA. People in the treatment group were offered a self-management programme and those in the control group received standard care. Participants were assessed at baseline, pre-intervention (6 weeks), post-intervention (12 weeks) and follow-ups (6 and 12 months). Assessments included self-report questionnaires, tender joint score and blood tests.

Following the self-management intervention, the treatment group showed significantly greater sustained improvements in individualized quality of life, self-efficacy for pain and other arthritis symptoms and use of planning as a coping strategy, when compared to the control group.

In the pooled sample, baseline illness representations were associated with coping procedures in the predicted directions. Perceiving RA to have multiple incoherent symptoms and serious consequences was associated with less use of adaptive and greater use of maladaptive coping strategies. Perceiving RA to be more controllable was associated with greater use of adaptive coping strategies.

Cross-sectional analyses, at baseline, showed that Illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social function and disease activity).

In the longitudinal analyses, baseline illness representations and coping procedures predicted a significant amount of variance in future health outcomes (pain, disability, emotional distress, disease activity and health care utilization) at 3, 6 and/or 12 months. Changes in illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social function, occupational function, individualized quality of life and disease activity). Changes in illness representations and coping procedures were stronger and more consistent predictors of health outcomes than baseline illness representations and coping procedures.

Illness representations mediated the improvements in individualized quality of life and self-efficacy for pain and other arthritis symptoms found immediately following self-management intervention. Self-efficacy for pain and pain coping strategies moderated the maintenance of treatment gains in individualized quality of life and self-efficacy for other arthritis symptoms respectively.

Results are discussed in relation to the published literature, methodological limitations are described, theoretical and clinical implications delineated and areas for future research identified.

Chapter 1: The challenge of Rheumatoid Arthritis

1.1 Introduction

This chapter explains the clinical context for the study. The clinical features, epidemiology, pathogenesis, course and prognosis of rheumatoid arthritis (RA) are reviewed first. Next the impact of RA on the person, health services and society are described. Finally current biomedical approaches to treatment and their limitations are discussed as a background to the rationale for psycho-educational intervention.

1.2 Clinical features of RA

RA is a chronic systemic autoimmune disease characterized by a symmetrical inflammatory polyarthritis. Cardinal features include unpredictable episodes of severe joint pain and swelling, stiffness, fatigue, and restricted function in activities of daily living. It often begins in the small joints of the hands and feet. Although initially the number of joints involved may be small it tends to spread to other joints over time. The joints most commonly affected are in the fingers, hands, wrists, toes, feet, ankles, shoulders, elbows, hips and knees. Inflammation of the synovium erodes articular cartilage and underlying bone. This destruction of the joints frequently leads to permanent deformity and progressive disability. Systemic features may include ocular, respiratory, cardiac, gastrointestinal, renal, neurologic and haematologic manifestations.

There is no simple definition of RA. It is actually a family of related diseases rather than a single entity. Diagnosis is difficult especially in the early stages of the disease when it may follow a variable and non-specific course. In the absence of an obligate pathognomonic test

diagnosis relies on a combination of clinical observations and laboratory investigations. A number of criteria have been proposed for identification and classification of RA. In 1958 the American Rheumatism Association proposed classification criteria for RA (Ropes, Bennett, Cobb, Jacox & Jessar, 1958) which were revised in 1987 (Arnett *et al.*, 1988), (see Appendix A).

1.3 Epidemiology

1.3.1 Epidemiology of Arthritis

There are over 200 types of arthritis and related conditions but RA is the most common form of inflammatory joint disease. The results of two recent UK epidemiological studies commissioned by the Arthritis Research Campaign show that Arthritis represents a significant, serious and widespread problem (Arthritis Research Campaign, 2002). These studies showed that almost twice as many people believe they have arthritis as report their condition to their General Practitioner (GP). The first study, a MORI Poll, estimated that 13 million people would report that they are currently affected by arthritis and joint pain. The second study, a review of available epidemiological statistics conducted by the ARC Epidemiology Unit, estimated that 7.1 million UK adults experience long-standing ill-health due to a musculoskeletal problem (Office for National Statistics, 2002).

Research on the epidemiology of RA has been reviewed by Hazes and Silman (1990), Symmons and Bankhead (1994) Silman and Hochberg (2001) and most recently by the ARC Epidemiology Unit (ARC, 2002)

1.3.2 Incidence of RA

Using the 1958 ARA criteria for classifying 'probable' and 'definite' RA, the Mayo clinic in Rochester, USA reported an overall annual incidence rate between 1965-1974 of 67 per

100 000 population with rates of 40 per 100 000 and 83 per 100 000 in males and females respectively. When only cases with 'definite' RA were included, the rates decreased to 22 and 48 respectively. The sex ratio was 1:2.5 and the incidence rates increased with age in both sexes (Linos, Worthington, O'Fallon & Kurland, 1980).

In the UK the latest incidence data has come from the Norfolk Arthritis Register, which is based on GP notifications in the Norwich Health Authority. It estimated an annual incidence of RA in 1990 of 36 per 100 000 for women and 14 per 100 000 for men (Symmons, Barrett, Bankhead, Scott & Silman, 1994). Overall, there are around 12,000 new cases of RA in the UK each year (Symmons *et al.*, 1994). There is no evidence that the incidence of RA differs throughout the UK.

1.3.3 Prevalence of RA

RA is very common. The prevalence in the US has recently been estimated as 10 per 1000 (1%), affecting 2.1 million people, 600 000 men and 1.5 million women (Arthritis Foundation, 1999). Most studies of the prevalence of RA in European populations have suggested an overall prevalence estimate for 'definite' RA of around 0.8% (Symmons & Bankhead, 1994). In the UK Lawrence (1977) reported a prevalence rate of 1.1% for 'definite' RA. The prevalence rate for 'probable' and 'definite' RA combined was 4.3%. The most recent UK prevalence figures come from the Norfolk RA prevalence Survey 2000, and show that around 387,000 adults in the UK have RA. This is 0.81% of the adult population (Symmons *et al.*, 2002).

There is a marked sex difference; the ratio (men: women) of people with RA is 1:2.7. The prevalence of RA rises steeply with age. The breakdown of adults with RA in the UK by age is as follows: young adults (aged 16-44): 17 000; middle-aged adults (aged 45-64): 158 000. Older Adults (aged 65+): 212 000. The ratio of young: middle-aged: old is 1:9:12 (ARC, 2002). The breakdown of adults with RA by country within the UK is as follows: England: 323 500; Scotland: 33 100; Wales: 20 500; Northern Ireland: 9 600 (ARC, 2002). There are some variations in the prevalence of RA by ethnic group. The

prevalence of RA is lower in people of Pakistani and African Caribbean origin than in European Caucasians (Silman & Hochberg, 2001)

1.3.4 Epidemiological trends

Reviews of the epidemiological evidence on RA suggest that from the 1950s there was a decline in both the incidence and prevalence of RA. The decline was most notable in women and for the very elderly RA is now more common in men than in women (Symmons & Bankhead, 1994). Over the last ten years it is likely that the number of people in the UK with RA has fallen. However directly comparable figures are not available because the definition of RA has changed. There are competing influences at play. The prevalence of RA rises quite steeply with age and so, as the population ages, the number of people with RA rises. However, during the 1970s-1980s the incidence of RA in women fell. This was probably due to a protective effect of the oral contraceptive pill. Incidence rates have been steady for the last 10 years. However, the effect of the fall in incidence two decades or so ago is still working through with regards to the prevalence (ARC, 2002). RA may also be becoming less severe with successive generations of patients being less likely to be seropositive, erosive or to have nodules (Silman, Davies, Currey & Evans, 1983)

1.4 Pathogenesis of RA

The specific aetiology of RA is unclear. It is a multifactorial condition and many different factors probably play a role in different individuals. Epidemiological evidence implicates genes (Brown & Wordsworth 1998; Ollier & MacGregor, 1995). RA tends to cluster in families and more women than men are affected. Whilst RA is not directly inherited, there is a familial association with the disease. Certain tissue types appear to influence susceptibility or course of the disease (Ravinder & Feldmann, 1998).

There are a number of non-genetic risk factors for RA. These include smoking (Albano, Santana-Sahagun & Weisman, 2001) and obesity (Silman & Hochberg, 2001). Some cases

of RA appear to be triggered by common infections or by childbirth. An infectious aetiology for RA has been extensively studied, but attempts to identify a specific agent have failed. It is likely that the risk factors for RA act in a cumulative fashion (Silman & Hochberg, 2001)

Although many of the details of the initiation and progression of RA remain unclear, a general picture has emerged that suggests that presentation of an as-yet unknown antigen results in clonal expansion of antigen stimulated T cells in a genetically susceptible individual during the early stages of the disease (Panayi, Lanchbury & Kingsley, 1992; Yocum, 1999). A small subset of activated T cells in the synovium has been shown to produce a variety of cytokines, including interferon- γ (IFN- γ), interleukin (IL)-2, IL-6 and tumour necrosis factor- α (TNF- α), that serve to sustain the inflammatory synovitis characteristic of RA (Smolen *et al.*, 1996; Steiner *et al.*, 1999). Further stimulation of other cell types in the synovium (dendritic cells, monocytes, B cells, fibroblast-like synoviocytes) either by cytokines or by direct cell contact with activated T cells, results in a second and more destructive stage of the disease. Activated monocytes and fibroblastic synoviocytes not only produce a variety of pro-inflammatory cytokines (particularly IL-1 and TNF- α) and growth factors that may perpetuate the inflammatory state, but also stimulate the production of matrix metalloproteinases and other proteases. It is these agents that mediate the breakdown of the tissue matrix of the joint characteristic of the destructive phase of RA (Firestein, 1996; Ivashkiv, 1996; Malik, Greenfield, Wahl & Kiener, 1996).

The specific mechanisms leading to the initiation and perpetuation of RA have not been identified but are an area of current active research (Edwards, 2002). It seems likely that inflammation in RA is mediated primarily by macrophage cytokine production. The potency of Tumour Necrosis Factor (TNF) neutralising agents supports the concept that TNF is the crucial cytokine, but others may play both parallel and amplifying roles. What has been more difficult to establish is the cause of macrophage activation. Although RA is sometimes seen as the conversion of an acute event into a chronic process, the weight of evidence is that RA is chronic from the outset. Histological studies indicate that the elements of early and late synovitis are much the same (Kraan *et al.*, 1998). Disturbances

of antibody production, alterations in immunoglobulins including rheumatoid factors, gather momentum for years before symptoms appear (Halldorsdottir, Jonsson, Thorsteinsson & Valdimarsson, 2000). The underlying process appears to involve an immune response with antigen presentation to T-cells via class II, often production of RF of all isotypes and macrophage cytokine generation (Edwards, 2002).

1.5 Course and prognosis of RA

The natural course of rheumatoid arthritis (RA) is characterized by increasing morbidity and excess mortality (Pincus & Callahan 1989). There are several studies with follow-up times of around 5 years, (Eberhardt & Fex 1998; Uhlig *et al.*, 2000; Young *et al.*, 2000). However, the long-term course, over 15 years, has been examined in only a few studies (Rasker & Cosh, 1984; Scott, Symmons, Coulton & Popert, 1987).

Symptoms of RA can start at any age but the peak age of incidence is in the early to mid 40's and it rarely occurs before puberty. Rheumatoid arthritis is extremely heterogeneous with regard to severity and rate of progression. The course and prognosis in individuals is difficult to predict. In the majority it runs a course of unpredictable exacerbations and remissions of inflammation with a gradual advance of destructive changes in the joints and moderate to severe disability. However the severity of the disease can range from mild self-limiting joint inflammation to progressive destruction of multiple joints with systemic manifestations.

Studies suggest that 5-10% of people with RA will eventually become very severely disabled by progressive disease, and around 20% will experience a remission of at least six months duration (Masi, 1983). Although early and permanent remission may occur in some cases, this is rare once permanent joint damage has started. However, this does not rule out clinical improvement after initially severely impaired function (Eberhardt *et al.*, 1990).

RA generally manifests in three different patterns. The monocyclic type is characterized by a brief duration of no more than a few months, and minimal disability. The polycyclic type has periods of exacerbation and remission, and usually causes less disfigurement and disability. The chronic progressive type has slow but relentless progression, which can be very debilitating (Fries, 1995). In a five-year follow up of 183 people with definite RA, Eberhardt and Fex (1998) categorized 56% as relapsing remitting and 44% as chronic progressive. In clinical practice, however, features of both types are frequently present in many patients. An alternative distinction is that between widespread and more limited chronic joint involvement (Wollheim, 1998).

There are two major components to the rheumatoid process in the joint - inflammation or synovitis (which is potentially reversible) and erosive damage to the articular cartilage and underlying bone (which is essentially irreversible). The conventional explanatory paradigm for the development of joint damage and disability in RA is that persisting inflammatory synovitis leads to progressive joint damage that subsequently results in functional disability. Recent evidence, discussed below, suggests that inflammation, joint damage and disability may follow different courses and may not be as closely related as was originally thought.

Joint damage begins shortly after the onset of disease (Van Leeuwen *et al.*, 1994). For example, in a cohort of patients with symptoms of RA for less than one year who were evaluated prospectively, 70% demonstrated evidence of radiographic joint damage after only 3 years' follow-up (Van Der Heijde, Van Leeuwen, Van Riel & Van de Putte, 1995). Moreover, the rate of progressive damage during the first year of follow-up was significantly more than in the second and third years. Although most people show progressive joint damage, there is evidence that in some cases erosions can heal (Houssein, 1998; Rau & Herborn, 1996). In the long term joint damage progresses steadily over the course of RA varying between 1.6-1.9% maximal possible damage annually (Scott *et al.*, 2001). For example Wolfe and Sharp (1998) demonstrated in a longitudinal study of a group of RA patients over 19 years, an almost linear progression of joint damage (Wolfe & Sharp 1998). However there are marked individual differences

in the patterns of progression of joint damage for different joints and people (Scott *et al.*, 2001).

The predictors of joint damage have been examined in a number of studies. Joint damage at five-year follow-up of people with early RA was closely related to damage at baseline (Uhlig, Kvien, Haavardsholm & Smedstad, 1998). This was also found by Fex, Jonsson, Johnson and Eberhardt (1996) but not by Möttonen *et al.*, (1998). Joint damage at follow up was also predicted by baseline levels of systemic markers of inflammation (erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP)) (Fex, *et al.*, 1996; Kaarela, 1985, Plant, Saklatvala, Borg, Jones & Dawes, 1994; Uhlig *et al.*, 2000; Van Leeuwen *et al.*, 1994; Wolfe & Sharp, 1998) and RF-positivity (Kaarela, 1985; Plant *et al.*, 1994; Möttonen *et al.*, 1998; Van Schaardenburg *et al.*, 1993; Van Zeben, Hazes, Zwinderman, Vandenbroucke & Breedveld, 1993; Uhlig *et al.*, 2000; Wolfe & Sharp, 1998).

It has generally been assumed that in RA clinically evident synovitis causes erosions. The cumulative amount of inflammation and the cumulative amount of erosive damage are correlated (Borg *et al.*, 1988). However the relationship is far from perfect, since every Rheumatologist has patients with no evidence of synovitis and a normal ESR who continue to erode (Symmons, 1994). If synovitis causes erosion then there should be a strong relationship between synovitis and erosions within specific joints. In an elegant study Kirwan (1997) examined the relationship between clinical synovitis (joint swelling and tenderness) and the development of erosions over one year in hand joints. The correlation between synovitis and erosions for individual joints was low. When all the joints of the hand were taken together the correlation was higher although still modest. These results argue against there being a direct causal relationship between synovitis and erosions in RA. They suggest that erosions and the signs of synovitis both reflect underlying disease activity but are not caused by the same pathological mechanisms.

With respect to the development of disability, as mentioned above, a fundamental paradigm of RA is that disease activity acting over time causes structural damage which

in turn produces functional impairment. This is supported by clinical observation and by studies showing that longer disease duration is associated with increased mortality (Erhardt, Mumford, Venables & Maini, 1989; Mitchell *et al.*, 1986; Pincus *et al.*, 1984; Soderlin, Nieminen & Hakala, 1998; Wolfe *et al.*, 1994), work disability (Callahan, Bloch & Pincus, 1992; Jantti, Aho, Kaarela & Kautiainen, 1999; Mau *et al.*, 1996; Reisine, Fifield & Winkelman, 1998; Sokka, Kautiainen, Mottonen & Hannonen, 1999; Wolfe & Hawley, 1998; Yelin, Meenan, Nevitt & Epstein 1980), joint replacement (Wolfe & Zwillich, 1998), and radiographic damage (Drossaers-Bakker *et al.*, 1999; Wolfe & Sharp, 1998).

Longitudinal studies using the Steinbroker Functional Class show that the number of people with moderate/severe disability increases over time. For example 9.4% of patients with early RA (mean duration of symptoms of 8.1 months) had marked functional loss (Functional Class III/IV) at presentation, compared with normal function in 33%, and by 5 years these numbers had increased in each group, respectively, to 16% and 40% (Young *et al.*, 2000). By 15 years most patients show some functional deterioration. Rasker and Cosh (1984) found, for patients with RA of less than 5 years duration, that 15% were in Functional Class III/IV (moderate/severe disability). After 15 years, 40% of patients with RA were in Functional Class III/IV.

However, studies using self-report measures of functional disability suggest that the development of disability in RA is more complex (Kvien, 2002). The most widely accepted self-report measure of functional disability for people with RA is the Health Assessment Questionnaire (HAQ; Fries, Spitz, Kraines & Holman, 1980). A recent large-scale study of patients with RA followed up for up to 20 years (Wolfe, 2000) demonstrated a stable course of HAQ scores at a group level but also major individual variation in scores over time. HAQ disability scores were high at disease onset and increase over time only very slowly, on average 1% of the maximum possible disability score per year (Wolfe, 2000). Individual RA patients have differing characterizable disability courses: 1) non-linear, 2) chaotic, or 3) non– time determined. There are large

inter and intra-individual variations in disability scores over time (Kirwan, 2001; Wolfe, 2000).

These results were supported by Scott *et al.*, (2000). They reviewed published studies and unpublished data on the progression of joint damage, the progression of disability and their interrelationship. Joint damage and disability both increase throughout the course of RA. The average annual rate of progression in HAQ disability scores, from the 9 studies reviewed, is low at 1% of the maximum HAQ score (0.031 units/year). This is remarkably similar to that reported by Wolfe (2000) who found an increase of 0.03units/year. Scott *et al.*, (2000) also reported marked individual differences in the patterns of progression of disability. Disability (HAQ score) was correlated with disease duration (correlation coefficients between 0.27 and 0.30) but the relationship was weak. This confirms Wolfe's (2000) finding that disease duration only explained 5% of the variance in HAQ scores. In summary, the model that physical disability, as measured by the HAQ, occurs as a function of disease acting over time does not fit the data well. Wolfe (2000) proposes that a biopsychosocial model provides a better explanation for the development of disability in RA. There is now considerable evidence that a wide range of factors are important in the development of disability and that different factors are likely to be important for different individuals and at different points in the disease process.

1.6 Health outcomes in RA

Adverse outcome is the suffering or loss of health experienced by an individual as a result of the process of disease. Outcome in RA has been described mainly in terms of mortality, radiographic damage (discussed above), and functional disability scores (Fries *et al.*, 1980). Only recently have psychological and socio-economic dimensions been considered, for example in the development of the American College of Rheumatology (ACR) and EULAR core data sets (Young *et al.*, 2000). RA frequently leads to early mortality, distressing symptoms, functional limitations, depression, work disability and socio-economic deprivation. However there are large individual differences in the health status of people with RA. People who appear to have similar levels of disease activity and

severity may have markedly different health outcomes. The health status of people with RA and factors that have been associated with health outcomes in RA will be considered. The role of psychological factors in predicting health status in RA will be described further in the next chapter.

1.6.1 Mortality

National mortality statistics for the year 2000 attributed 790 deaths in the UK to RA. (Office for National Statistics, 2000; General Register Office for Scotland, 2000; Northern Ireland Statistics & Research Agency, 2000). National mortality data probably underestimate real mortality due to RA because RA is unlikely to be recorded on the death certificates, particularly as the underlying cause of death (Office for National Statistics, 2000; United Kingdom Health Statistics, 2001).

Most studies conducted on survival and death in RA indicate a reduction of life expectancy in RA patients (Cosh, 1984; Spector & Scott, 1988). The median life expectancy was shortened by 7 years in males and 3 years in females (Vandenbroucke, Hazevoet & Cats, 1984). People with RA have a risk of early mortality that is double that of demographically matched groups from the general population (Wolfe *et al.*, 1994). The increased mortality is mainly due to infections, renal disease, respiratory disease, gastro-intestinal complications of therapy and rheumatoid arthritis itself (Pincus & Callahan, 1986)

The main loss of life expectancy occurs in patients with more severe disease (Rasker & Cosh, 1981, Mitchell *et al.*, 1986, Vandenbroucke *et al.*, 1984) and could for the most part be attributed to the disease itself (Rasker & Cosh, 1981 and Vandenbroucke *et al.*, 1984). Apart from disease severity, increased mortality is associated with early onset RA (Rasker & Cosh, 1981) and the presence of LE cells (Vandenbroucke *et al.*, 1984). Pincus *et al.*, (1984) showed in their original studies that self reported disability predicted survival in RA. These results were later confirmed by a longitudinal analysis of a patient sample from the 1980s (Callahan *et al.*, 1997). Wolfe *et al.*, (1988) found a two-fold increase in mortality rate in patients having moderate disability (HAQ score between 1 and 2) and a

four- to five-fold increase in mortality in patients with HAQ scores between 2 and 3. Individual patients with RA at risk for early mortality may be identified by the presence of a number of indicators including many involved joints, co-morbid cardiovascular disease, and poor functional status (Pincus & Callahan, 1993a).

1.6.2 Symptomatology

People with RA frequently experience a variety of distressing and disabling symptoms including pain, stiffness, fatigue and sleep disturbance.

1.6.2(a) Pain

Pain is usually ranked as the most important symptom for people with RA (Gibson & Clarke, 1985). Being free of pain is the primary concern for patients with RA (Deyo, 1988). The quality of pain experience in arthritis was investigated by Melzack (1975). On the McGill Pain Questionnaire the majority of patients with arthritis used words like aching, exhausting and rhythmic to describe their pain. More than a third mentioned gnawing, annoying, and constant. Wagstaff, Smith and Wood (1985) report that patients with arthritis use terms such as throbbing and burning and not terms such as scalding, drilling and cutting. The quality of pain is also dependent on the nature of the activity the person is doing. In RA pain at rest and pain while moving are qualitatively different. Pain at rest is described as throbbing or aching and pain while moving is described as shooting and spreading (Papageorgio & Badley, 1989).

Clinical measures of disease activity, physical examinations and current methods of imaging have in general not been found to predict the extent of pain in RA (Merskey, 1996). Parker *et al.*, (1988a) found that ESR, Ritchie Articular Index, grip strength, walking speed and morning stiffness were only able to account for 3% of the variance in pain scores. However demographic and social factors, such as age and income were reasonable predictors of pain.

In RA there are strong reciprocal relationships between pain, disability and depression (Huyser & Parker, 2002). The association between pain and disability has been examined in a number of studies. In early RA joint pain was correlated with functional disability (Van Leeuwen *et al.*, 1994). In established RA, Parker *et al.*, (1988a) found the extent of pain was related to physical disability measured using the Steinbroker Functional Classes (Steinbroker, Traeger & Batterman, 1949). They report that people with RA classified as Functional Class III (functional capacity quite limited) reported much more pain on a visual analogue scale of pain intensity and a greater area of their body was painful when compared to those classified as Functional Class I and II. Pain has also been found to be associated with functional disability assessed on self-report measures, such as the HAQ (Ward & Leigh, 1993).

There is increasing recognition that pain in RA is a multidimensional phenomenon. On a physiological level, pain sensation and inhibition is related to an interplay of central nervous system and peripheral mechanisms (Crofford & Casey, 1999). Cognitive, emotional, behavioural and social processes play an important role in shaping the experience of pain in RA. Higher pain levels have been associated with psychosocial variables like lower self efficacy, increased distress, decreased social support, and decreased coping skills (Bradley & Alberts, 1999; Buescher *et al.*, 1991; Newman, Fitzpatrick, Lamb & Shipley, 1990).

Relationships between pain and psychosocial variables can be complicated. For example, emotional disorders like depression may cause pain, or, conversely, chronic pain may predispose individuals to experience depression. Also, the relationship between anxiety and pain appears to be curvilinear, where anxiety is apt to increase pain if an individual is at rest, while high levels of anxiety in the face of danger (e.g., a soldier in the midst of battle) will decrease perceived pain levels (Merskey, 1996). In any case, regardless of the hypothesized direction of the association, these examples highlight the critical ways that psychosocial variables can affect the experience of pain.

Although there is a strong relationship between pain levels and psychosocial variables in RA, pain does have, at least in part, a significant physiologic basis (Dessein, Shipton &

Budd, 2000). A greater understanding of such physiologic pathways may help illuminate psychosocial aspects of pain in rheumatic disease. There may be dual physiologic pathways for pain and rheumatologic disease activity, thus accounting for the relative disjunction between pain and disease activity (Affleck *et al.*, 1997).

There also may be a physiologically based predisposition for individuals with chronic pain to experience negative affective states like anger, depression, and anxiety, without the clear mediation of psychosocial factors. Fernandez and Milburn (1994) suggest that in response to aversive stimuli like pain, humans are "physiologically prewired" to experience negative affect, which in turn motivates adaptive processes. For example, they argue that anger and anxiety motivate flight/fight responses, while depression motivates submission responses. This notion is supported by the identification of a direct sub cortical pathway through which pain receptors transmit afferent signals to the thalamus and the amygdala, which are responsible for releasing anger responses (LeDoux, 1987).

1.6.2(b) Fatigue

Fatigue is a common symptom of RA. Estimates of the prevalence and severity of fatigue vary. More than 80% of people with RA experience at least some fatigue (Belza, 1996; Wolfe, Hawley & Wilson, 1996). Clinically significant levels of fatigue were reported by 41% of people with RA (Wolfe *et al.*, 1996). Although RA does not include fatigue as a diagnostic criterion some people with RA regard fatigue as their most severe symptom (Fitzpatrick, Newman, Jenkinson & Mowat, 1992). As many as 57% of people with RA have indicated that fatigue is the most problematic aspect of their disease (Tack, 1990). The significance of fatigue is emphasized by the finding that it is an important predictor of work dysfunction and general measures of health status (VAS of global severity, health status, and health satisfaction) (Wolfe *et al.*, 1996).

In addition to the subjective experience, fatigue can be conceptualised as having both objective physiologic (e.g., increased lactate levels) and behavioural (e.g., decreased physical and mental performance) dimensions (Huyser & Parker, 2002). However, in

clinical populations objective measures of fatigue have not been unambiguously correlated with subjective measures, thus, most studies of fatigue in RA focus on subjective fatigue (Piper, 1989).

Wolfe *et al.*, (1996) found that fatigue was correlated with demographic variables and measures of disease activity in RA. However, in multivariate analyses they found that disease activity (assessed by ESR, joint count and grip strength) was not associated with fatigue. Wolfe *et al.*, (1996) found the strongest independent predictors of fatigue were pain, sleep disturbance, depression, tender point count and disability (HAQ). Other studies in RA have confirmed that fatigue is most strongly associated with psychosocial variables like higher levels of pain and depression, and lower levels of social support (Huyser *et al.*, 1998; Riemsma *et al.*, 1998).

1.6.2(c) Sleep

High frequencies of sleep disturbance have been found in RA. Pincus, Swearingen, and Wolfe (1999) found, in a large sample of people with a variety of rheumatic disorders, that 75% had problems getting a good night's sleep. The mean scores for sleep problems in the RA group were similar to those for most of the other rheumatic disorders. People with RA often experience sleep fragmentation (i.e., light easily disrupted sleep with multiple awakenings). This disturbance has been associated with increased disease activity (Crosby, 1991).

In their study, Pincus, Swearingen, & Wolfe (1999) found that sleep problems were not strongly associated with age, disease duration or educational level but were strongly associated with HAQ disability, pain, fatigue and depression.

Pain is the most commonly cited cause of sleep disturbance among individuals with rheumatic conditions (Wegener, 1996). In a longitudinal study of RA, pain predicted future sleep disturbance (Nicassio & Wallston 1992). These relationships are likely to be bi-directional and poor sleep may produce increases in pain (Huyser & Parker, 2002) and

fatigue (Wolfe *et al.*, 1996). Finally, increased rates of depression and anxiety in people with RA also may contribute to sleep problems. Conversely sleep disturbance may contribute to the development of depression (Nicassio & Wallston 1992).

1.6.2(d) Joint stiffness

Joint stiffness, especially in the morning, is a characteristic feature of RA. However it has received little research attention and it is not clear that it can be distinguished from pain. The subjective qualities of stiffness are not well understood. Rhind, Bird and Wright, (1980) found, in 100 patients with rheumatological conditions, that spontaneous descriptions of joints included the term "stiffness", which was described as limitations in movement. Early morning stiffness, which frequently lasts for several hours, often necessitates major adjustments in lifestyle.

1.6.3 Physical disability

Arthritis and related conditions are the leading cause of disability in the US (Kelsey, 1982) and the UK (Badley & Tenant, 1993). The 1988 OPCS survey estimated that three million people in the UK had a disability associated with musculoskeletal disorders (Martin & White, 1988). Over half of the population who are over 85 years had joint problems and most of these reported some disability (Badley & Tenant, 1993). RA is one of the most severely disabling forms of arthritis. Although population studies show that OA and back pain are the most common cause of disability in rheumatic disease, people reporting RA have the most current symptoms, make more use of medical services and have the highest level of disability (Badley & Tenant, 1993). In a large scale study of hospital outpatients with a variety of rheumatic diseases, Pincus *et al.*, (1999) found that patients with RA had higher physical disability scores, on a modified HAQ, than people with other rheumatic diseases.

As discussed above, long-term follow-up studies using the Steinbroker Functional Class Show that functional deterioration occurs in most patients surviving 15 years or longer

(Rasker & Cosh, 1989). Disability levels measured using the HAQ are high throughout the course of RA and increase slowly over time (Wolfe, 2000). Epidemiological studies show that significant physical disability is common in RA. For example, Kvien *et al.*, (1997) found, in an area in which the prevalence of RA was 0.5%, that about half of the population of people with RA (up to 80 yrs of age) had a Modified Health Assessment Questionnaire (MHAQ) score exceeding 1.5. This score indicates severe disease and a high risk of reduced life expectancy and future additional disability (Wolfe *et al.*, 1988). In the same area, the incidence of RA with a MHAQ score exceeding 1.5 after 5 yrs was 10/100 000, with an MHAQ score exceeding 2 the incidence was 3.5/100 000 and with an AIMS2 physical score exceeding 4 it was 4/100 000 (Uhlig, Kvien, Glennås, Smedstad & Førre, 1998).

RA can affect a very wide range of functional activities. Inflammation of the joints and tendons results in pain, swelling and restricted movement. These difficulties are exacerbated by progressive joint damage and deformities. People with RA have reduced joint flexibility, muscle strength and endurance and aerobic capacity that leads to restricted performance of functional activities (Van den Ende, Vliet Vlieland, Munneke & Hazes, 1998). Mobility difficulties, for example, problems with walking, bending, climbing stairs, reaching, stretching and dexterity, are common in RA (Fries *et al.*, 1980; Meenan, Gertman & Mason, 1980; Wolfe *et al.*, 1988). People with RA also have difficulties engaging in many activities of daily living, for example toileting, washing, dressing, preparing meals, housework and shopping (Fries *et al.*, 1980; Meenan *et al.*, 1980; Wolfe *et al.*, 1988). RA also affects people's ability to continue valued activities such as driving, social, sporting and leisure pursuits (Katz, 1995; Meenan *et al.*, 1992; Pincus *et al.*, 1999). In RA, where there is a preponderance of women, it has been suggested that the ability to perform domestic roles and activities is particularly important. Reisine, Goodenow & Grady (1987) found that many women with RA report limitations performing domestic activities, for example cleaning the house (73%), doing laundry (65%), shopping (61%). RA also affects the ability to perform nurturing roles such as giving attention and support to other household members and maintaining family ties with others outside the home. Allaire, Meean and Anderson (1991) found that women with mild RA spent as much time on

housework as women without RA but achieved less. Women with more severe RA spent less time on housework than those with mild RA but their families spent more time on housework. Current and possible future problems of mobility and loss of independence are a major concern for people with RA (Burckhardt, Archenholtz & Bjelle, 1993). The clinical significance of disability, assessed on self-report measures, is emphasized by the finding that it is an important predictor of future functional disability, work disability, joint replacement surgery and mortality (Wolfe, 2000).

As discussed above, duration of RA is only weakly associated with self-reported disability. There is evidence that radiological damage is an important predictor of HAQ in established RA. Scott *et al.*, (2000)'s review found that the link between X-ray damage and disability is stronger (correlation coefficients between 0.30 and 0.70) than that between disease duration and disability. In the earliest phases of RA, X-ray damage and HAQ scores were not related, but by 5–8 yrs there were significant correlations ($r=0.3-0.5$). In late RA, greater than 8 years, most studies show highly significant correlations ($r=0.3-0.7$) and so in late RA (>8 years) joint damage accounted for 25% of variance in disability. They concluded that initially patients have little radiological damage but considerable disability, as a consequence of having active disease. Over the next 3-8 years radiological damage increases and this results in increased disability. The link between joint damage and disability is strongest in late RA. However, avoiding or reducing joint damage in both early and late RA is likely to maintain function.

In addition to joint damage a number of other factors have been shown to be important predictors of disability. A variety of demographic factors influence HAQ scores. These include age (HAQ increases with age; Anderson *et al.*, 1988), low socio-economic and educational status (HAQ is higher in the poor and under-educated; McEntegart *et al.*, 1997; Vliet Vlieland *et al.*, 1994), sex (HAQ is higher in women; Thompson & Pegley, 1991) and income (relative poverty is associated with high HAQ scores; Wolfe *et al.*, 1988).

A number of measures of disease activity and inflammation are associated with HAQ disability. Higher HAQ is associated with rheumatoid factor positivity (Van Zeben *et al.*, 1993; Woolf *et al.*, 1991), especially IgA rheumatoid factor (Houssein, Jonsson & Scott, 1997). Measures of systemic inflammation, such as ESR, are associated with HAQ scores (Wolfe 2000). Systemic inflammation is an important predictor of disability in early RA, Devlin *et al.* (1997) and Smedstad *et al.*, (1996) found high levels of CRP and ESR were related to higher HAQ scores. However this relationship between HAQ scores and inflammation remains fairly constant throughout the course of RA. A chronic progressive pattern of disease activity is associated with higher HAQ when compared to a relapsing remitting pattern Suarez-Almazor, Soslone, Saunders, Russell, (1994) Eberhardt and Fex (1998). Genetic factors including DR4 have been shown by some (Gough *et al.*, 1994, Van Zeben *et al.*, 1991 and Moreno *et al.*, 1996), but not all, studies (Eberhardt, Fex, Johnson & Wolheim, 1996) to be positively associated with high HAQ scores. Receiving specialist rheumatological care and therapy with slow-acting and other anti-rheumatic drugs is associated with lower HAQ disability (Fitzgerald & Bresnihan, 1983; Fries, Williams, Morfeld, Singh & Sibley, 1996; Porter, McInnes, Hunter & Capell, 1994; Ward, Leigh & Fries, 1993). Scott *et al.*, (2000) also note that other factors, such as muscle strength and the involvement of large joints, might be expected to influence disability but there is insufficient evidence.

Baseline level of HAQ is one of the best predictors of future HAQ (Wolfe 2000, Scott *et al.*, 2000). Pain is also strongly associated with higher HAQ scores (Ward & Leigh, 1993) and fatigue is closely related to both pain and disability levels (Wolfe *et al.*, 1996). There is also evidence that Psychological factors (e.g. depression) are important predictors of disability in RA (Katz & Yelin, 1993; McFarlane & Brooks 1988; Newman, Fitzpatrick, Lamb, & Shipley, 1989; Wright *et al.*, 1996).

Overall, the predominant determinants of HAQ disability in RA are baseline HAQ, systemic inflammation, joint damage, pain and psychological factors (Scott *et al.*, 2000; Uhlig *et al.*, 2000; Wolfe, 2000). It appears that inflammation contributes much more to

the level of disability during the first years of the disease course, whereas joint damage contributes more strongly after about 10 yrs of disease duration (Kvien 2002).

1.6.4 Emotional distress

Over the past 20 years, it has become increasingly clear that RA not only produces substantial physical disability but also has important psychological consequences (Escalante & Del Rincon, 1999; Van der Heide *et al.*, 1994; Yelin & Callahan, 1995). Given that RA leads to early mortality, unpredictable and distressing symptoms, joint deformities and disfigurement, functional disability and restricted ability to participate in social roles, it is not surprising that it may lead to difficulties in emotional adjustment (e.g. depression, anxiety, lower self esteem and reduced life satisfaction). In RA depression and depressive symptomatology have been most extensively investigated. The overlap between symptoms of RA and the somatic symptoms of depression can cause difficulties in the diagnosis of RA and in the identification and treatment of depression in RA (Creed & Ash, 1992).

Prevalence estimates for depression and anxiety in RA vary considerably (e.g. 14-42%; Huyser & Parker, 2002). In a review of several large-scale surveys of clinic and community samples, DeVellis (1993) concluded that levels of depression and depressive symptomatology are higher in people with RA than for people without chronic illnesses, but are similar to those found in people with other chronic illnesses. As RA is one of the most serious rheumatic diseases it might be expected that there would be higher levels of emotional distress in RA than in other rheumatic diseases. However, in a large 10-year study, Hawley and Wolfe (1993) found that levels of depression and anxiety in RA, assessed with self report measures, were not significantly different from those for people with most other rheumatic diseases attending hospital clinics. Similar results have been reported by Pincus *et al.*, (1999). However, a study using a semi-standardized psychiatric interview, found that a greater number of people with RA (23%) than people with OA (10%) met diagnostic criteria for depression (Abdel-Nasser *et al.*, 1998). In RA the prevalence of depression is higher in hospital out patient clinic samples than in

community samples (DeVellis, 1993). A review of studies based on self-report questionnaire measures of depressive symptomatology reveals great discrepancies in estimated prevalence, ranging from 20-80% (Pincus *et al.*, 1996). Estimates of the prevalence of depressive disorders in RA, which are based on semi-standardized psychiatric interviews, are lower (e.g. 7-21%; Creed & Ash, 1992).

Reduced positive affect has also been found in RA. In an analysis of three cross-sectional studies of RA, Zautra *et al.*, (1995) report that increased negative affect and reduction in positive affect are significant and independent consequences of RA.

Higher prevalence rates for clinically significant levels of anxiety have also been found in RA in a number of studies (Frank & Hagglund, 1996; Pincus *et al.*, 1999). Clinically significant levels of anxiety may even be more common than those for depression. In a UK sample, using the Hospital Anxiety and Depression Scales, Pincus, Griffith, Pearce, & Isenberg, (1996) report a prevalence rate for depression of 23% compared with 6% in case controls, using the 8 plus cut-off to indicate possible clinical depression. The prevalence rate was 15% using the more stringent 11 plus cut-off for probable depression. Unfortunately they do not report a prevalence rate for anxiety. However, the mean anxiety score (mean=8.13; SD=4.8) was higher than the mean depression score (6.22, SD 3.8). In addition, the odds ratio showed that RA patients were twice as likely to be depressed and 4 times as likely to be anxious as case controls. There is little empirical research on other emotional responses to RA, such as anger and frustration, although clinical reports suggest that these may be important (Huyser & Parker, 2002).

With respect to the course of emotional distress, several longitudinal studies have shown that prevalence rates for early and established RA remain relatively stable over time (Hawley & Wolfe, 1993; Katz & Yellin, 1993; Uhlig *et al.*, 2000). For example, Hawley and Wolfe (1993) found stable AIMS scores for anxiety and depression in 400 patients over an average of 3.1 yrs. However, at the individual level there is considerable variation in emotional distress over time. A longitudinal study by Katz and Yelin (1993) showed that 15%– 17% of people with RA had depressive symptoms each year and that

depression in any given year substantially increases the likelihood of depression in subsequent years. However, only 4% of participants had depression throughout the study and 29% experienced depressive symptoms during the study. Overall, although many people will experience episodes of significant emotional distress the majority make a reasonable emotional adjustment (DeVellis, 1993; Barlow, 2001a).

When depression does occur in RA it is particularly significant because it is a strong predictor of poorer physical health outcomes (e.g. days spent in bed; DeVellis, 1993) and greater use of health-care services (Frank & Hagglund, 1996). Katz and Yelin (1993) found that persons with RA who were depressed had significantly poorer physical function and generally reported more RA- related physician visits and hospitalisations. Depression and anxiety can also heighten pain and increase disability in RA (Keefe & Bonk, 1999; Yelin & Callahan, 1995). Beckham *et al.*, (1992) found that depression predicted a moderate to large amount of the variance in physical and psychosocial disability, even after analyses controlled for important demographic and medical status variables. Depression is one of the strongest predictors of physical disability in RA (McFarlane & Brooks, 1988). In their study of early RA Uhlig *et al.*, (2000) found that the 5-yr change in physical function (AIMS) was predicted by emotional status (AIMS) at baseline.

Many studies have examined the relationships between emotional distress and other relevant variables. The determinants of depression in RA are complex and multifaceted. Psychological distress is often only weakly related to demographic factors, disease activity and objective indicators of disability.

With respect to demographic factors, age has been found to be associated with depression in RA. Wright *et al.*, (1998) found that younger people with RA were significantly more likely to report depressive symptoms on the CES- D than older people. In a study of 110 persons having RA, Devins, Edworthy, Guthrie, and Martin (1992) found that as the lifestyle disruptions of RA become more intrusive, younger people were much more likely to develop depressive symptoms than older people. However, an interesting study

by Shifren, Park, Bennett, and Morrell (1999) found that older people with RA who had difficulty with cognitive tasks tended to report much lower self-efficacy, higher pain, and worse mental health outcomes.

Some studies have found that measures of disease activity are associated with depression. Abdel-Nasser *et al.*, (1998) found in RA that erythrocyte sedimentation rate was significantly associated with clinical depression. Parker *et al.*, (1992) found that individuals with higher counts of painful or swollen joints were more likely to report depressive symptoms on the BDI. However, most studies have failed to find associations between markers of the activity and severity of RA and the extent of depression (Peck, Smith, Ward & Milano, 1989). Although associations have been found in some cross-sectional studies, longitudinal studies have found that demographic and clinical measures of disease activity and severity do not predict emotional distress over time, for example over an average 3.1 years in established RA (Hawley & Wolfe, 1988) and over five years in early RA (Uhlig *et al.*, 2000).

Research suggests that emotional distress is more strongly associated with other variables, for example pain and functional disability (Frank & Hagglund, 1996). However, the relationships between emotional state, pain and disability are complex and bi-directional. Pain is associated with depression in RA (Creed & Ash, 1992) but, as discussed above, the direction of causality is unclear. Brown (1990) suggests a causal model in which pain predicts subsequent depression. Nicassio and Wallston (1992) extended this work in a longitudinal study of 242 people with RA. Prior pain predicted adverse changes in sleep problems and the interaction between high sleep problems and high pain were independently associated with depression. Conversely, Parker *et al.*, (1992) found depression had more influence over pain than vice versa. There is also evidence that a history of depression before the onset of RA predicts future pain. Fifield, Tennen, Reisine, and McQuillan (1998) randomly selected a sample of 203 persons with RA from a national panel and conducted a telephone interview to assess their history of major depression, current depressive symptoms, and pain, fatigue, and disability. The authors excluded any individual who currently met criteria for major depression. Participants who

reported more depressive symptoms at the time of interview had higher levels of pain. More important, this study found participants who had a previous history of major depression were more likely to currently have higher levels of pain, even if the episode of depression occurred prior to the onset of RA.

Scores on self-report measures of physical function, such as the HAQ, are associated with levels of depression (Abdel- Nasser *et al.*, 1998; Peck *et al.*, 1989). The loss of the ability to perform valued activities seems to be an important predictor of depression. Katz and Yelin (1995) conducted a longitudinal study of persons having RA in which they examined how declines in function were related to depression (Geriatric Depression Scale Short Form). Although overall level of functional decline did not predict depressive symptoms, the loss of valued activities was a significant predictor of the subsequent development of depressive symptoms. Depression may also influence perception of disability. Higher levels of depression could produce a downward bias in perceived ability to perform daily activities. Longitudinal studies are needed to disentangle the precise nature of causal relationships between pain, depression and disability.

Finally, there is increasing evidence, discussed further in Chapter 2, that psychosocial variables (e.g. prior depression, daily stressors, illness related cognitions, coping strategies, and social support) appear to be better predictors of anxiety and depression than demographic variables or measures of disease activity (Bradley & Alberts, 1999; Wright *et al.*, 1996).

Overall, a significant minority of people with RA experience clinically significant levels of emotional distress. Depression in RA is especially important because it can exacerbate symptoms and functional disabilities, and lead to worse health status. There is some evidence that demographic factors and clinical measures of disease activity may be associated with psychological distress. However, other factors like pain, functional ability, and psychosocial variables appear to be more important in the development of depression. Early identification and aggressive treatment of depression may be particularly important because it could prevent and reduce the impact of depression on arthritis pain, disability

and health-care utilization (Parker & Wright, 1995). Psychological factors implicated in the development of depression are of interest as they may be amenable to treatment and contribute to the prevention of future depression.

1.6.5 Work disability

Rheumatic disease is the leading cause of work loss and the second leading cause of work disability payments in the U.S. (Allaire, 1996). RA has a dramatic impact on occupational function. The fastest decline in employment rate is within the first three years of disease onset (Mau *et al.*, 1996). RA affects work performance even soon after diagnosis. Doeglas *et al.*, (1995) found, in patients with a mean time since diagnosis of 2 years, that 90% had given up work or made changes in their work because of RA. Young *et al.*, (2000) found, in their UK study of early RA, that at the onset of RA 48% were in paid employment. After 5 years, 40% of these people were no longer working. Of the 40% who stopped working, 13% stopped work either because they had reached their normal retirement age or for other reasons, such as redundancy, and for the other 27% RA was the main or a contributory reason for stopping work. Within five years of disease onset 50% of RA patients, who worked before the onset of RA, stop work (Yelin *et al.*, 1980) and after 10 years 60% had stopped work (Pincus & Callahan, 1993b).

There is evidence that workforce participation for men with arthritis is declining in the US. For men aged 55-64 years old, between the 1970s and 1980s, participation declined from 54% to 38% (Yelin, 1992). However, younger women with arthritis have increased their workforce participation (Yelin, 1992). Lower workforce participation in people with arthritis has a significant effect on their incomes. For people with arthritis, men had 48% and women 27% of the income of those without arthritis (Mitchell, Burkhauser & Pincus 1988). Workforce participation may also affect health status. In a study of 723 hospital outpatients with RA those without paid employment had higher levels of pain and depressed mood even when controlling for disease severity Fifield, Reisine and Grady (1991).

Pain, fatigue, limited physical functioning, uncertainty, extensive treatment demands and vulnerability to psychological distress can be challenges that the individual with arthritis has to overcome when trying to enter or maintain their position in the workplace. Younger people with arthritis report a variety of difficulties in employment including lack of awareness, confidence and support (Straughair & Fawcitt, 1992).

People with arthritis face a number of attitudinal, environmental and organizational barriers in society (Barlow & Harrison, 1996). Arthritis is generally viewed as a condition associated with old age. This attitude derives from a lack of understanding in society and is a constant source of tension for younger people with arthritis (Barlow, Shaw, & Harrison, 1999b). People with invisible or fluctuating arthritis are often viewed as being lazy, malingering, or using their condition to vindicate their failure to meet the social expectations of others. Environmental barriers frequently derive from the inappropriate design of physical infrastructures that effectively limit, not only access to buildings, but also freedom of movement once inside. Access to transport and buildings can prove to be major barriers encountered by people with arthritis in their attempt to enter or maintain employment.

A number of risk factors for work disability have been identified. Poor disease status is a predictor of work disability for people with RA (Allaire, 1996). However, remaining in employment is better predicted by social characteristics of the work (e.g. being able to control the pace of work and being self employed) than medical variables (Yelin *et al.*, 1980). The most important issues are the amount of flexibility of and control over work that people have, enabling people with RA to control the pace and timing of activities at work. Doeglas *et al.*, (1995) found an association between educational level and remaining in employment despite RA. This was interpreted as reflecting the nature of work performed by those with a higher educational level. They found that 32% of non-manual workers and 80% of manual workers with RA had left their jobs. Not surprisingly therefore people in professional or managerial occupations are more likely to retain employment Callahan, Bloch and Pincus (1992). Other psychosocial variables also appear to be involved. For example, significant risk factors for RA-related work

disability include less social support and depression (Allaire, 1996). Overall RA frequently leads to work disability that in turn affects health and socio-economic status. Men in manual occupations are at significant risk of work disability.

1.7 The cost of RA

The number of people with arthritis in the UK is rising as the population ages, and as a result, the problem is becoming an increasing drain on the NHS and on society. Arthritis and related conditions are the second most common cause of days off work in both men and women, after mental disorders (ARC, 2002). In England for one year (1992), the economic impact of disability associated with RA has been estimated at £1.256 billion and is mostly accounted for by lost production (McIntosh, 1996). In 1999-2000 9.4 million working days were lost because of RA in Great Britain. This represents £833 million lost production. (Department of Work & Pensions, 2001). The economic burden on individuals is also significant. For example, people with RA experience a 50% drop in their income over a 9- year period (Doherty, Brandt, & Lohmander, 1998).

The management of RA makes major demands on health and social care resources. There were 1.9 million GP consultations for inflammatory arthritis in 2000 (Office for Health Economics, 2001). There were 45,887 hospital admissions for inflammatory arthritis in 1999-2000 (Department of Health, n.d.). The costs of medical care for people with RA are three times the costs of those for age and gender matched individuals who do not have RA (Allaire, Prashker, & Meenan, 1994) and are similar to those of persons having coronary heart disease (Lubeck, 1995). The estimated direct costs of arthritis and related conditions to health and social services was £5.5 billion (ARC, 2002). Hip and knee replacements cost £405 million (ARC, 2002). Although arthritis is a chronic, growing problem, NHS expenditure appears to be going in the opposite direction. Between 1990 and 1999, NHS expenditure on arthritis and related conditions increased by 5%, compared with an increase of 19% in the total NHS budget (ARC, 2002). This means that as the problem grows, the share of the budget being applied to it appears to shrink. The

prevalence and severity of problems related to arthritis, and the costs associated with it, necessitate new approaches to tackling the problems.

1.8 Treatment and management of RA

There is no known cure for RA. The emphasis of treatment is on reducing inflammation, relieving symptoms (pain, stiffness and fatigue), preventing joint damage and optimising physical function. Typically, treatment is long term and complex comprising medication, exercise, use of equipment (e.g. splints) and joint replacement surgery. Treatment is accompanied by the need for long-term monitoring, often involving regular attendance at hospital or general practitioner clinics.

Pharmacological treatment for RA aims to reduce inflammation in order to control symptoms and to slow down the progression of the disease. This is important, because the longer the disease persists, the more the joints get damaged. Once damaged, they heal very poorly, so it is vital to prevent the damage occurring.

The first line of drug treatment in RA is usually Non-Steroidal Anti-inflammatory Drugs (NSAIDs). These are used to reduce the signs and symptoms associated with inflammation (e.g. joint swelling and tenderness). Many types are available, and in a small number of people this is enough to manage the disease. However for most people RA remains active despite optimal treatment with NSAIDs and they require "second line drugs" such as gold, Methotrexate and steroids. These Disease Modifying Anti-Rheumatic Drugs (DMARDs) are used to suppress the underlying inflammatory process and slow progression of joint damage and disability. In the ERAS study 84% of patients were prescribed at least one DMARD by 5 years (Young *et al.* 2000). In recent years there has been an increasing realisation of the importance of early diagnosis and effective treatment to prevent joint damage and disability in RA (Bresnihan, 2002). The lag times between onset of symptoms of RA and diagnosis, and the introduction of disease modifying anti-rheumatic drugs, have been greatly reduced.

Several new drug therapies have recently been introduced for the treatment of RA. These include the cyclooxygenase-2 inhibitor celecoxib and the new DMARD leflunomide. A new class of drugs, targeted biological agents, that block or interfere with the function of cytokines involved in the pathogenesis of RA have also become available (e.g. the anti-tumour necrosis factor agents, etanercept and infliximab). New drugs are also being developed that are targeted against components of the immune activation and co-stimulatory pathways. These include antibodies against the interleukin-1 (IL-1) receptor and blockers of the CD28 and CD40 co-stimulatory pathways (Simon & Yocam, 2000). These new treatments have the advantage of better side effect profiles or providing additional options for limiting early joint damage and subsequent disability in patients who have not responded to other drug therapies (Simon & Yocam 2000).

Pharmacologic approaches can in some instances significantly reduce symptoms of the disease such as pain and disability. However in many cases pharmacotherapy is only partially effective in relieving symptoms, reducing disability and preventing disease progression. For example medication only produces a 20-50% improvement in symptoms (Hirano, Laurent & Lorig, 1994). The use of pharmacotherapy is also limited by loss of efficacy and side effects. For example, Pincus, Marcum and Callahan (1992) found that in most cases DMARDs were discontinued after less than 2 years either because of loss of efficacy or serious adverse events. NSAIDs and DMARDs frequently produce serious side-effects (e.g. gastrointestinal complications and compromised immune system function) particularly with prolonged use (Astin, Beckner, Soeken, Hochberg, Berman, 2002). As noted by Gotzsche (1989) such adverse effects can in some instances become so problematic that patients either become noncompliant, or opt for drugs with fewer side effects, even if these medications are less effective. Many people are left with distressing symptoms, disabilities, and uncertainty about the future course of their illness despite optimal pharmacotherapy.

Specialist hospital care is generally provided by a multi-disciplinary team (MDT). The MDT may include: Rheumatologists; orthopaedic surgeons; Rheumatology nurse specialists; physiotherapists; occupational therapists; podiatrists; social workers;

dieticians and psychologists. Vliet Vlieland and Hazes (1997) reviewed 35 studies comparing in-patient and outpatient MDT care with routine care. There was evidence that MDT care produced greater benefits in disease activity, physical function, psychological well-being and social adjustment, at least in the short term. (Vliet Vlieland & Hazes 1997).

Therapists (Physiotherapists, Occupational therapists and Podiatrists) offer a range of treatment modalities aimed at improving and facilitating daily functioning. For example Young *et al.* (2000) found that 83% of early RA patients were seen by a therapist during the first five years.

Physiotherapists can offer exercise therapy, which is considered to be a cornerstone of the treatment of RA in all stages of the disease. Exercise therapy is aimed at preserving and restoring overall functional ability by improving joint mobility, muscle strength and aerobic capacity. Traditionally, the most important objectives of exercise therapy were to preserve joint mobility and to maintain muscle strength. Exercise forms, which put little stress on the joints, like range of motion exercises and non-weight bearing, isometric exercises were advocated (Baker, 1953; Jivoff, 1975; Swezey, 1974). Previously, dynamic exercises with intensity adequate to improve muscle strength and aerobic capacity were thought to exaggerate pain and disease activity and to provoke joint damage. During the last two decades dynamic exercises, to increase muscle strength and aerobic capacity, have been recommended, in particular for people with RA without active disease (Hicks, 1990; Semble, 1995; Sutej & Hadler, 1991). These recommendations are supported by a systematic review of four methodologically sound studies of dynamic exercise for RA (Van den Ende *et al.*, 1998). They found that it was effective in increasing aerobic capacity and muscle strength. No detrimental effects on disease activity and pain were observed.

Occupational therapists can provide specific treatments, such as training in joint care/protection (Hammond, Lincoln & Sutcliffe, 1999) and the use of orthotics (Henderson & McMillan, 2002; Huyser & Parker, 2002). They also help people with

major adaptations and aids. In Young *et al.* (2000) study of patients with early RA, within 5 years of disease onset 10% of patients needed major adaptations or appliances that included stair adaptations, stair lifts, major bathroom and toilet changes, or regular use of wheelchairs. A further 18% of patients required walking aids (special shoe wear and callipers). Joint-replacement surgery has traditionally been used in people with established RA where a major joint is badly damaged and painful. In recent years surgery for people with early RA and a wider range of joints has become routinely available. The ERA study found that of 732 people with early RA, followed for five years, 117 (17%) underwent orthopaedic surgery for RA, 55 (8%) for major joint replacements (Young *et al.*, 2000).

Given the chronic nature of the disease, the persistence of symptoms and disability despite treatment, the side effects associated with many pharmacologic approaches and the recognition of the psychosocial impact of RA, there is growing awareness of the need to incorporate psychosocial approaches into ongoing biomedical treatment for managing RA (Simon & Yocam, 2000). These issues are discussed further in Chapter 3.

1.9 Summary

RA is the most common form of inflammatory arthritis, with a peak age of incidence in the early to mid forties. The causes of RA are poorly understood. The disease is chronic, progressive, incurable and the prognosis is uncertain. It follows an unpredictable course of exacerbation and remission of inflammation with gradual progression of joint damage. RA is associated with excess mortality, distressing symptoms, moderate to severe functional disability, increased risk of depression and major social and economic consequences. The costs of RA to the person, health service and society are enormous. Current bio-medical approaches to treatment are ameliorative aiming to reduce inflammation, relieve pain and maintain or improve function. However they lack prolonged efficacy, often produce side-effects and frequently fail to address the psychosocial needs of people with RA.

Chapter 2: Psychological processes in the course and outcome of Rheumatoid Arthritis

2.1 Introduction

Over the past 30 years there has been considerable research interest in the role that psychological factors may play in the development, course and outcome of RA (Anderson, Bradley, Young, McDaniel & Wise, 1985). This chapter reviews evidence related to what Anderson *et al.*, (1985), have termed "the disease course hypothesis". This proposes that psychological factors play a significant role in the process of adaptation and eventual outcomes in established RA.

Bio-medical models, such as the World Health Organisation ICIDH (WHO, 1980) and ICIDH-2 (WHO, 2000) suggest that the pain and disability associated with RA should be directly related to disease activity and severity. However in RA there is considerable evidence that the relationship between measures of disease activity and severity and pain, disability and emotional distress is more complex (Newman, 1998). Objective measures of disease severity and activity (radiographic measures of joint damage and biochemical markers of auto-immune activity) explain little variance in pain (Merskey, 1996; Parker *et al.*, 1988a), disability (McFarlane & Brooks 1988a; Uhlig *et al.*, 2000) and emotional distress (Hawley & Wolfe, 1988; Peck, Smith, Ward & Milano, 1989).

Psychosocial factors have been shown to be much stronger predictors of these health outcomes in RA (Huyser & Parker, 2002). One difficulty in interpreting these results is the fact that the test-retest reliability for psychological factors tends to be significantly higher than phasic indicators of disease activity, such as Erythrocyte Sedimentation Rate (ESR) or C-reactive protein (CRP). However, despite fluctuations in activity, one would expect that when disease activity is measured at the same time as health outcomes such as disability, that there would be considerable association between the measures. Rather, the

association between psychosocial factors and health outcomes is a well-replicated result, which usually accounts for a greater proportion of the variance in self-reported pain, disability and depression than physical variables (Young, 1992). Fries and Belamy (1991) suggest that a biopsychosocial model of health may be more helpful for understanding the pain and disability associated with rheumatic disease than the traditional bio-medical model.

2.2 The biopsychosocial model

Engel (1977) first proposed the biopsychosocial model and argued that it could be used to understand how persons cope with a variety of diseases. The biopsychosocial model continues to be seen as a very useful approach to understanding both pain (Keefe & Bonk, 1999) and disability (Fries & Belamy, 1991; Osterweis Kleinman, & Mechanic, 1987). As shown in Figure 2.1, a biopsychosocial model maintains that the pain and disability experienced by people with RA is not only affected by underlying biological factors but also by psychological and social factors (Keefe *et al.*, 2002).

"The biopsychosocial model provides a systems perspective on arthritis. Changes in one part of the system can produce changes in another part of the system. For example, increases in disease activity (a biological change) can lead to increases in anxiety and depression (psychological changes) and decreases in the ability to work or perform household chores (social changes), both of which, in turn, can increase pain and disability. Alternatively, improvements in a person's self-efficacy about controlling arthritis symptoms (a psychological change) can lead to enhanced compliance with medications (producing biological changes) or increased interaction with supportive friends and family (a social change), both of which can reduce pain and disability."

(Keefe *et al.*, 2002)

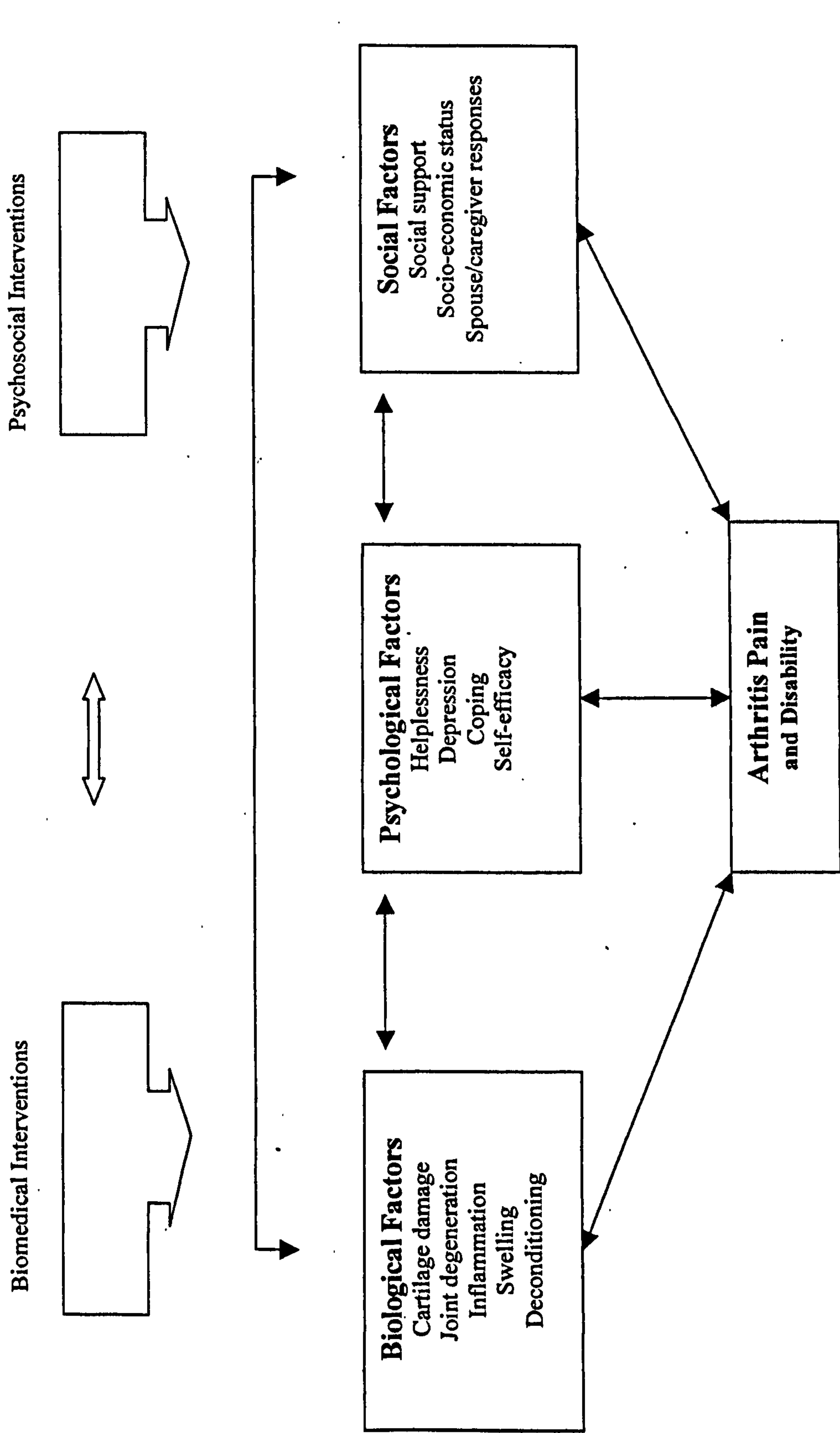


Figure 2.1: The biopsychosocial model of arthritis pain and disability. Adapted from Keefe and Bonk, 1999.

As shown in Figure 2.1, the biopsychosocial model can be used not only to understand arthritis pain and disability but it can also serve as a guide for psychosocial interventions. Treatment protocols that target psychological and/ or social factors can potentially modify the arthritis experience. Recent controlled studies (reviewed in Chapter 3) have shown that biopsychosocial interventions can be useful in reducing arthritis- related pain, disability and depression.

Biopsychosocial research in arthritis has drawn on a range of specific psychological theories, models and frameworks. For example perceived control (Wallston, 1993), learned helplessness and hopelessness (Abramson, Seligman & Teesdale, 1978; Alloy, Abramson, Metalsky, & Hartlage, 1988; Seligman, 1975), stress and coping (Folkman & Lazarus, 1986), self- efficacy (Bandura, 1977) and social support (Revenson, 1993).

2.3 Leventhal's self-regulatory model

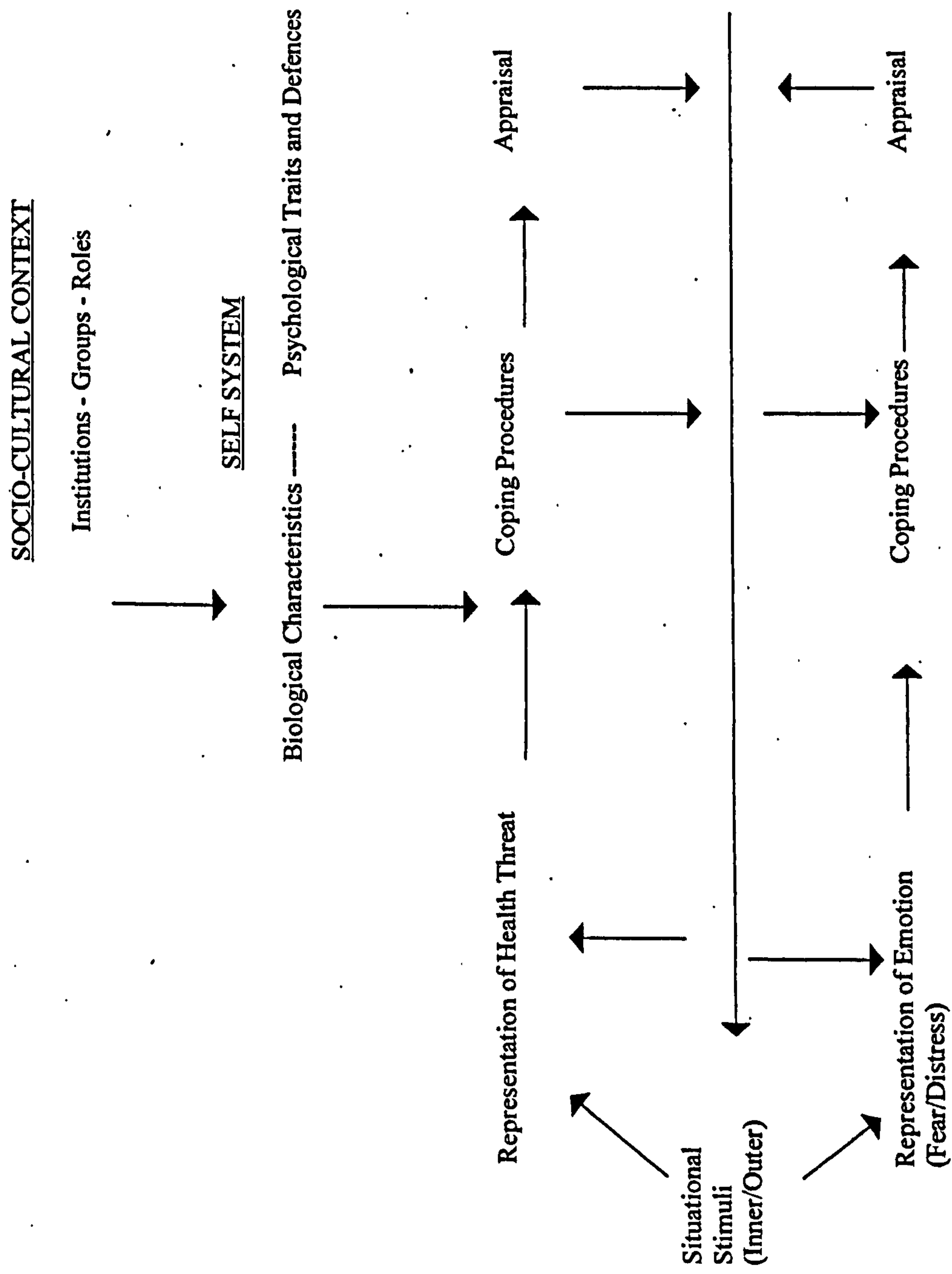
The main focus of the current study is on the application of one biopsychosocial approach, Leventhal's Self-Regulatory Model (SRM; Leventhal, Meyer & Nerenz, 1980; Leventhal, Nerenz & Steele, 1984; Leventhal, Diefenbach & Leventhal, 1992a; Leventhal, Leventhal & Shaefer, 1992b). Leventhal's SRM is one of the most influential theoretical approaches which has guided research exploring how people adapt to illness over the past 20 years. This framework enables integration of many of the specific theories and models that have been applied in this field. Research guided by related approaches will be discussed and integrated within this framework where appropriate.

Leventhal's SRM provides a framework for understanding the processes by which people make sense of and respond to health threats (see Figure 2.2). The SRM suggests that people actively construct representations of illness experience by evaluating health threats against implicit common sense models of illness. This has been likened to the search to find a meaning in adverse life situations (Schiaffino & Cau, 1995).

The SRM hypothesises that people create mental representations of their illness based on the concrete and abstract sources of information available to them in order to make sense of and manage the problem. It is the interpretation of this information that forms the first step in the process of seeking help, engaging in a coping strategy or adopting an illness management regimen (Bishop 1991).

The illness representation is based on three basic sources of information (Leventhal *et al.*, 1980; Leventhal *et al.*, 1984). The first source of information is the general pool of lay information already assimilated by the person from previous social communication and cultural knowledge of the illness. The second source is information from the external social environment, from perceived significant others or authoritative sources (e.g. a partner or doctor) Finally, the person completes their illness representation by taking into account their previous and current experiences with the illness. Personality and socio-cultural factors may also be important in this process (Diefenbach & Leventhal, 1996). The cognitive and emotional aspects of illness representations, occurring in response to a health threat, determine the nature of coping procedures, and the appraisal of the effectiveness of coping.

Figure 2.2: Leventhal's Self-Regulatory Model (SRM; adapted from Leventhal, Diefenbach & Leventhal, 1992a).



Research has consistently shown that the cognitive representation of illness has at least five main attributes.

Identity - which includes the label the person uses to describe their illness and the signs and symptoms that they view as being part of the illness such as pain and fatigue; *Cause* - the perceived causes of the illness and exacerbations/remissions; *Time line*- whether the illness is expected to be acute, episodic or chronic; *Consequences* - the expected effects and outcomes of the illness such as loss of independence; *Cure or Control* - beliefs about the extent to which the illness is amenable to cure or control.

Cognitive representations of illness mediate between the signs and symptoms of the disease and emotional responses and coping procedures. The self-regulatory processes are dynamic and feedback from appraisals of coping efforts in turn influence cognitive representations, emotional responses, and coping efforts (Leventhal *et al.*, 1980, 1984, 1992a, 1992b). According to this framework the outcome of the adaptation process is primarily determined by the illness representation that people develop, subsequent coping efforts and appraisals of coping.

In their review, Petrie and Weinman (1997) report that support for the SRM has been found in a wide range of acute and chronic illnesses, for example hypertension (Meyer, Leventhal & Guttman, 1985) and diabetes (Hampson, Glasgow & Toobert, 1990). Early research focused largely on relating illness representations to coping procedures, such as compliance with medication. However later work has shown that there is considerable evidence that self-regulatory processes influence broader health outcomes such as pain behaviour, activity limitations work disability and psychosocial adjustment (Johnston, 1996; Scharloo *et al.*, 1999).

2.4 Application of the self-regulatory framework to RA

A number of researchers have argued that Leventhal's SRM may be particularly helpful in understanding and managing rheumatic disease (Hampson, Glasgow & Zeiss, 1994; Park,

1994; Pimm & Weinman, 1998; Scharloo *et al.*, 1999; Schiaffino & Revenson, 1992; Sharpe, 1999). With no cure for RA and with medical treatments only being partially effective it is important to explore the self-regulatory processes that mediate between the disease, pain, disability, and psychological adjustment. Understanding how some people successfully manage RA, minimizing pain and disability and optimizing psychological adjustment may help in the development of interventions to assist people who are managing relatively poorly.

2.5 Psychosocial processes in adaptation to rheumatic disease

Over the past 30 years, numerous studies have explored the role of psychosocial processes in the course and outcome of RA (Anderson *et al.*, 1985; Keefe *et al.*, 2002). These studies indicate that psychological processes such as perceived control, helplessness, self-efficacy, depression, stress, and coping are important in understanding differences in how persons with arthritis respond to their disease. Social factors, such as social support and socio-economic status, are also increasingly being shown to be important in understanding arthritis pain, disability and emotional well-being (Shipley & Newman, 1993). Many of these studies are particularly salient, because they have led to new assessment and treatment protocols for managing arthritis. Research on processes that have particularly important implications for the biopsychosocial management of RA is reviewed below. These psychosocial factors can broadly be grouped into four themes which are encompassed within Leventhal's SRM: cognitions; coping procedures; appraisals of coping; social context. These four areas of research will be reviewed and linked to the SRM.

2.5.1 Illness related cognitions and appraisals

A number of studies have investigated the features of RA that people appraise as stressful. Van Lankveld, van't Pad Bosch, van de Putte, Naring and van der Steak (1994) found that people with RA perceived the most stressful aspects of their illness to be pain, physical limitations and dependence on others. Perceptions of pain, limitations, and dependence while being only weakly related to assessments of disease activity and severity were

strongly related to measures of quality of life even after controlling for the objective measures.

Katz (1998) examined the impact that participants reported for different stressors related to RA. While many studies of stress and coping have traditionally asked only about pain, Katz's (1998) sample of 446 participants, rated fatigue, functional impairment and taking care of RA as equally difficult. Interestingly, they found that there was a correlation between coping efficacy and the impact that participants reported and these in turn were related to depressive symptoms.

Mahat (1997) also investigated different illness-related stressors and their relationship to coping efficacy. Their smaller sample, of 53 people with RA, did report pain as the worst stressor. However, as in Katz's (1998) work, they found that other stressors including limited mobility, inability to perform household tasks, dependency on others, social restriction, effects on family, work problems and discomfort arising from treatments were also rated as stressful. Mahat (1997) also found that participants rated the efficacy of different types of strategies as more or less effective depending upon the stressor that they were dealing with.

In the context of the SRM this evidence suggests that specific aspects of people's illness identity (e.g. pain and fatigue) and consequences (e.g. perceived limitations and loss of independence) are very salient features of people's illness representation. Further these illness representations are associated with other self-regulatory processes and health outcomes in RA.

Other illness-related cognitions have also been investigated, for example causal attributions. Affleck, Pfeiffer, Tennen and Fifield (1987a) found that people reported a variety of causes for their RA (heredity 34.7%, auto-immune factor 24.4%, personal behaviour 22.8% psychological stress 22.8%). Specific causal attributions were not related to functional ability or psychological adjustment, but those who were unable to identify any cause for their RA were more helpless and had poorer functional ability and psychological

adjustment. They also investigated attributions for the cause of symptom flares (psychological stress 45.5%, changes in weather 34%, excessive physical activity 34.1%) and remissions (changes in medication 49.4% and absence of psychological stress 21%). Attributing the cause of current disease activity to psychological stress was associated with greater functional disability, greater disease severity, and poorer psychosocial adjustment. Attributing the cause of flares to excessive physical activity was associated with less helplessness, whereas attributing the cause of remissions to changes in medication was linked with greater helplessness. This may suggest that attributing the cause of current disease activity to areas over which one has some personal control may be more adaptive. It is interesting to note that few people report attributions about the cause of remissions that would support a sense of personal control. In general this study suggests that in people with established RA, attributions about the cause of current disease activity play a greater role in adaptation to RA than attributions about the cause of the disease.

The concept of perceived control has also received considerable attention in the RA literature.

"Perceived control [PC] is defined as the belief that one can determine ones own internal states and behaviour, influence one's environment, and or bring about desired outcomes."

(Wallston, Wallston, Smith & Dobbins, 1987).

Early studies examined Locus of Control beliefs using questionnaires such as the Multidimensional Health Locus of Control scale (Wallston, Wallston, Kaplan & Maides, 1976). Felton and Revenson (1984) found that people with RA are as likely as people with cancer to believe that they have little personal control over their health, and hold more external locus of control beliefs than people with other chronic illnesses such as diabetes and hypertension.

Wallston (1993) reviewed the literature on perceived control in rheumatic disease. He found that in cross-sectional studies a strong internal health locus of control was

associated with better health outcomes (e.g. life satisfaction and quality of life) but in longitudinal studies it frequently failed to predict improved health outcomes. He suggests that for people with a chronic illness a strong internal locus of control may not always be the most adaptive illness representation. For example, Roscam (1986) showed that people who have high internal and powerful others and low chance/fate locus of control report less depression over time than people with other patterns of locus of control beliefs. Wallston (1993) concludes that, in influencing health outcomes in rheumatic disease, locus of control is not as important as the perception that health status can be controlled. There is evidence that greater perceived control over RA is associated with more favourable health outcomes including pain and disability (Flor & Turk, 1988) and depression (Roscam, 1986). Greater perceived control is also associated with higher use of active problem focused coping strategies and less use of passive and emotion focused coping strategies (Schussler, 1992)

The relationship between perceived control and health outcomes may not be as simple as these results suggest. As Leventhal's SRM proposes, other factors, such as the nature of the illness and feedback from evaluations of the effectiveness of coping efforts, may also be an important determinant of the adaptive significance of the illness representation. These points are illustrated in a study by Affleck, Tennen, Pfeiffer, and Fifeld (1987b) who conducted semi-structured interviews with 92 RA patients to investigate the relationship between disease status, more specific perceptions of control and predictability and psychological adjustment. The results indicated that individuals' perceptions of control differed for different aspects of the illness. People believed they had more control over daily symptoms than the course of RA, and viewed health professionals as having more control over the course of their illness than themselves. People who believed they had more control over symptoms and the course of RA also viewed RA as more predictable. There was evidence that, for people with more severe RA, perceiving personal control over relatively more controllable aspects of rheumatic disease and perceiving that powerful others have control over relatively uncontrollable aspects is more adaptive. Specifically they found that perceiving greater personal control over daily symptoms was associated with better positive mood in people with moderate or severe RA. Perceiving greater personal control

over the course of RA was associated with negative mood in people with severe RA. The importance of participation in treatment by people with RA is emphasized by their finding that perceiving greater personal control over treatment is associated with better mood, and perceiving greater health professional control over daily symptoms is associated with negative mood but greater perceived predictability. This result confirms the importance of concepts such as control and predictability, but illustrates the complex interactions between health status and self-regulatory processes.

In a further development of this work Affleck, Tennen, Urrows, and Higgins (1992a) studied daily reports of pain, activity limitations, and mood over 75 days in people with RA. They found that initial perceived control over pain was positively associated with decreased pain over time. The dynamic nature of self-regulatory processes in adaptation to RA is emphasized by the finding that pain reports moderated the relationship between initial perceived control beliefs and mood. People who initially assumed they had control over daily pain and then experienced increased pain became more distressed over time.

2.5.2 Coping procedures

Coping can be defined as people's cognitive and behavioural efforts to manage external or internal demands (Lazarus, 1993). Coping in RA refers to the methods that people develop to cope with the demands of the illness. Coping behaviours may be broadly divided into adaptive ways of coping assumed to be associated with better outcome versus maladaptive ways assumed to be associated with poorer response. Additional distinctions that have emerged in the coping literature include the difference between problem- versus emotion-focused coping, and passive versus active coping. There is considerable evidence to suggest that people with RA do develop ways to cope with their symptoms and that for many they develop very adaptive coping strategies independently (Keefe *et al.*, 1991).

Much of the literature on coping with RA has focused on the use of coping strategies to manage pain. Pain-coping strategies are the cognitive and behavioural efforts that

individuals use to cope with, deal with, or minimize their pain (Rosenstiel & Keefe, 1983). Cognitive pain-coping strategies can include such efforts as the use of distraction, prayer, or calming self-statements. Behavioural strategies to cope with pain often involve increasing or decreasing activity. There is considerable evidence that pain-coping strategies can be reliably assessed in people with persistent pain, and that these strategies are predictive of pain, psychological distress, and physical disability (Jensen, Turner, Romano, & Karoly, 1991; Keefe, Salley, & Lefebvre, 1992). For example, in a longitudinal study, Brown, Nicassio and Wallston (1989a) used the Pain Management Inventory (Brown & Nicassio, 1987) to investigate the role of active and passive pain-coping strategies in adjustment to RA. They studied 287 people with RA at two time points six months apart. The results of the cross-sectional analysis showed that pain, passive coping and an interaction of the two all contributed independent variance to the level of depression experienced. The longitudinal analysis showed that participants who engaged in higher levels of passive coping, when experiencing severe pain, were more likely to become severely depressed over time. Active coping efforts were also found to predict lower depression. The authors suggest that the association between passive coping and depression, in the face of severe pain, might be similar to a state of learned helplessness.

Some studies have explored how people cope with RA in general. Revenson and Felton (1989) looked longitudinally at the relationship between coping efforts, disability and psychological adjustment in RA. Their measure of coping was derived from the Ways of Coping Scale (Folkman & Lazarus, 1980). Although this is a general measure of the ways people cope with life, Revenson and Felton (1989) specifically added “in reaction to being ill”. They found that people with RA most commonly used wish-fulfilling fantasies, threat minimization, and cognitive restructuring to cope with their illness. A higher reported use of any strategy was related to heightened positive affect six months later. In addition, initially low levels of acceptance of RA and high levels of depression were associated with subsequent increases in disability. These findings indicate that coping efforts are associated with increases in positive mood over time. Mood and acceptance are subsequently related to disability.

Newman, Fitzpatrick, Lamb and Shipley (1990) examined differences in the patterns of coping behaviour used by people to manage chronic RA. Using cluster analysis they classified people into four groups. Group 1 used denial, avoidance of others during pain, reorganising routine, and seeking support from friends; Group 2, the largest group, had a passive pattern of coping, not strongly adopting/rejecting any coping strategy; Group 3: had the most open and active coping pattern, confronting their disease, refusing to reorganise routines, engaging in physical activity, and expressing feelings; Group 4 used rest, diet, religion, and prayer. People in group 3 who had an open and active coping pattern reported less pain and stiffness, physical disability, and better emotional well-being. The groups did not differ on demographic variables, laboratory or clinical measures of disease and it is unclear what factors account for differences in coping style. It is possible that cognitive representations of the illness or appraisals of coping efficacy may influence the development of different patterns of coping.

There is some evidence to suggest that people use different coping strategies to manage different illness stressors. Blalock, Devellis, Holt and Hahn (1993) conducted in-depth interviews to identify important life changes brought on by RA and to elicit information on how individuals coped with these changes. The interviews were transcribed, and a systematic content analysis was used to identify coping behaviours. The coping behaviours identified were then correlated with standardized measures of psychological and physical functioning. Data analysis revealed two interesting findings. First, the use of cognitive and behavioural strategies varied considerably across different problem areas, such as dealing with problems with daily activities versus dealing with problems with social relationships. Second, persons who showed very little flexibility in their coping behaviours were much more likely to have decreased psychosocial functioning.

Much of the research has been conducted with participants who have chronic RA. People newly diagnosed with RA may need to develop new coping strategies as methods they have used to cope with other stressors may no longer be appropriate or sufficient to manage the novel demands of RA. Evers and her colleagues have investigated the role of coping

strategies and social support with disability and mood, early in the disease. They investigated 91 patients recently diagnosed with RA at the point of diagnosis and one year later. With regard to emotional response, Evers, Kraaimaat, Geenan and Bijlsma (1997) found on cross-sectional analysis at the point of diagnosis, no relationship between coping and emotional distress. Even one year later, initial coping was unrelated to outcome on measures of psychopathology. Interestingly, however, relationships between current coping and level of distress did emerge in cross-sectional analyses after one year. In another investigation from the same cohort, Evers, Kraaimaat, Geenen and Bijlsma (1998) found that the level of functional impairment (assessed by a combination of both physical and self-report measures) could be predicted by frequent use of passive pain-coping strategies, particularly those of worrying and resting. These results seem to indicate that not only is coping important in chronic RA, but the strategies developed even very early in the course of the illness are related to outcome. It is interesting that in early RA coping appeared to have a stronger relationship to functional ability than distress.

Overall there is consistent evidence that coping behaviours play a significant role in adaptation to RA. Generally, passive, avoidant emotion-focused coping approaches (e.g. wishful thinking, self blame, expression of emotion) are associated with greater pain and psychological distress (Bradley & Alberts, 1999) and greater disability (Evers *et al.*, 1998). Active problem focused coping (e.g. rational thinking, information seeking, maintaining activity despite pain) are associated with positive affect and improved psychological adjustment (Young, 1992) and in some studies pain and disability (Newman *et al.*, 1990).

Although these studies offer consistent evidence of the relationship between coping, pain, mood and disability, the amount of variance accounted for by initial levels of coping in the studies cited is usually modest. This is perhaps not surprising given the long gaps between assessment and the number of extraneous measures that may contribute to changes in pain, mood and disability. In addition a problem with questionnaires and interview methods is that they rely on retrospective accounts of coping that may be

subject to recall bias (Stone, Kessler, & Haythornthwaite, 1991). Coping is considered to be a dynamic process that may not be adequately captured by questionnaire or interview measures that are administered at only one or a few points in time. To overcome these problems recent studies have used daily diary methodologies.

Affleck, Urrows, Tennen and Higgins (1992b) were among the first to use daily study methodologies to analyze the process of coping with RA. In their study, 75 persons having RA kept diary records of pain-coping, mood, and joint pain each day for 75 days. Pain-coping was assessed at the end of each day using Stone and Neale's (1984) Daily Coping Inventory (DCI) adapted for chronic pain coping. The coping strategies assessed by the DCI include

- (a) pain reduction effort,
- (b) relaxation,
- (c) distraction,
- (d) redefinition,
- (e) venting emotions,
- (f) seeking emotional support, and
- (g) seeking spiritual comfort.

Affleck *et al.*, (1992b) found that persons having RA were most likely to report the daily use of relaxation strategies and least likely to use the strategies of venting emotions and redefinition. They also found that people who used relaxation more frequently as part of their daily coping repertoire had less daily pain, and those reporting more coping efforts showed improved pain and mood over time.

Keefe *et al.*, (1997a) followed a cohort of 53 patients for 30 consecutive days. Patients made daily ratings of the frequency of coping, perceived efficacy of coping and of their pain levels and mood. Interestingly the cross-sectional design found the reverse relationship between coping and well-being to that usually reported in the literature. They found that the perceived efficacy of coping was unrelated to the subjective level of pain,

but greater use of coping strategies was positively associated with pain. While this may appear contradictory it seems likely that participants need to rely more on active coping strategies at times when the pain is severe. This interpretation is confirmed by the longitudinal results. They indicated that coping efforts were related to improvements in pain, but that these were on a time-lagged basis. That is, participants who relied heavily on active coping strategies and reported these to be efficacious subsequently reported lower levels of pain the following day. In addition, similar improvements in positive mood were also associated with coping the previous day. This study strongly supports the important mediating effect that coping tends to have on both mood and pain in RA. Further this study reveals the subtle dynamic nature of coping responses that occur in RA where symptoms, disability and mood may fluctuate from day to day.

Coping behaviours have received a great deal of research attention and there is evidence that they are related to pain, disability and emotional distress in RA. However, until recently, the links between coping and the cognitive and appraisal processes that provide the context in which coping behaviours are selected and evaluated have received less attention (Manne & Zautra, 1992; Newman & Revenson, 1993; Zautra & Manne, 1992). More recently studies have investigated the complex relationships between appraisals of illness and non-illness related stressors, coping efficacy, coping efforts and health outcomes.

2.5.3 Appraisals of coping efficacy

While coping strategies have been demonstrated to be important in mediating people's psychological and physical adjustment to RA, many of the recent studies have examined not only the frequency of use of coping strategies but also the perceived efficacy of those strategies. Efficacy is an interesting concept as it involves not only the knowledge of and ability to use coping strategies, but also involves the person's appraisal of their success. There is evidence that appraisals of coping efficacy play a significant role in self-regulation of rheumatic disease and are important predictors of health outcome. Three related psychological constructs have received attention in the literature:

- (a) catastrophizing,
- (b) self-efficacy
- (c) helplessness.

None of these constructs were originally conceived of as appraisals of coping efforts within Leventhal's framework, and it might be argued that these constructs could be conceptualised as coping procedures or cognitive representations of illness. However, broadly these constructs can be envisaged as cognitions and appraisals which the individual makes about their own coping responses.

2.5.3(a) *Catastrophising*

Catastrophizing was originally conceptualized as a method of cognitively coping with pain, characterised by negative self-statements and overly negative thoughts and ideas about the future. The patient unrealistically assumes that, given the current situation, the worst possible outcome will occur (Keefe, Brown, Wallston, & Caldwell, 1989). Alternatively catastrophizing could be viewed as a form of cognitive representation reflecting a particularly strong negative view of the identity of the disease and its consequences (Petrie, Moss-Morris & Weinman, 1995). However, some authors have argued that catastrophizing seems to reflect appraisals of coping efficacy (Parker *et al.*, 1989).

There is evidence that catastrophizing is an important dysfunctional process in the self-regulation of RA. Most of the research on catastrophizing in rheumatic disease has used the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983). This questionnaire assesses the use of coping strategies for pain and the extent to which the strategies are effective in the control or reduction of pain. One of the cognitive coping sub-scales assesses the use of catastrophizing. Keefe *et al.*, (1989) examined the role of catastrophising, using the subscale from the CSQ. They studied 223 patients with RA longitudinally. Their results demonstrated that levels of catastrophizing were stable over the study and the initial level of catastrophizing was significantly related to pain intensity, disability and



depression six months later. These relationships held even though the analysis controlled for medical status and the initial values on all dependent variables.

Keefe *et al.*, (1987) showed, in people with OA, that two factors from the CSQ, labelled "Coping Attempts" and "Pain Control and Rational Thinking" (PCRT), accounted for 60% of the variance in responding on the CSQ. The PCRT factor contains the catastrophizing sub-scale and the beliefs about pain controllability and coping efficacy items. Keefe *et al.*, (1991) investigated the PCRT in a cross-sectional study of people with RA. Higher scores on the pain control and coping efficacy items, and lower scores on the catastrophising subscale, were associated with better outcome, with regard to lower levels of pain and psychological disability. Following this several studies have investigated the coping attempts and PCRT factors in RA. Parker *et al.*, (1988b) used the CSQ to investigate the role of coping attempts and PCRT in adjustment to RA. They argued that Coping Attempts assessed frequency of strategy use, whereas PCRT assessed appraisals of coping efficacy. The study had a longitudinal design and in the year between the two periods of measurement all participants had completed a pain management programme. Significant changes over time were found in both the coping attempts and PCRT factors. Before treatment, the PCRT factor was related to lower levels of pain, less helplessness, less psychological distress and lower levels of stress (hassles). Furthermore, increases in the PCRT factor, over the year, were associated with decreases in the same variables. Coping attempts were directly related to pain scores, but not to psychological distress, helplessness or stress. They concluded that the confidence people have in their coping strategies is more important than the available coping resources in predicting adaptation in RA.

This work was replicated and extended in a later study. Wright *et al.*, (1996) investigated predictors of depression in a longitudinal study of 141 people with RA enrolled in a stress management intervention trial. A wide range of potential predictors was assessed including daily stressors, coping attempts and coping efficacy (assessed with the PCRT factor from the CSQ). The study demonstrated that depression could be predicted from a combination of daily stressors, lower levels of coping efficacy (PCRT) and a higher level

of physical disability. Coping attempts were not a strong predictor of depression. Overall, catastrophizing, and beliefs about pain controllability and coping efficacy have been shown to be important predictors of physical and psychological health outcomes in RA.

2.5.3(b) *Self-efficacy*

There is growing recognition that a related concept, self-efficacy, is an important variable in understanding how individuals adjust to arthritis (Barlow, Williams & Wright, 1996; Daltroy & Liang, 1993; Lorig, Chastain, Ung, Shoor, & Holman, 1989a; Lorig *et al.*, 1989b; Manne & Zautra, 1992; Parker & Wright, 1995). Self-efficacy has been defined as the belief that one has the ability to engage in a course of action sufficient to attain a desired outcome (Bandura, 1977).

Research on self-efficacy in arthritis was stimulated by the development of the Arthritis Self-Efficacy Scale (ASES) (Lorig *et al.*, 1989a). Studies show that people living with arthritis vary substantially in their self-efficacy (Lorig *et al.*, 1989a). Research has shown that higher levels of self-efficacy are associated with lower levels of pain, psychological distress, and functional impairment in people with arthritis (Lorig *et al.*, 1989a) and more specifically people with RA (Barlow, Cullen & Rowe, 2002a; Schiaffino, Revenson & Gibofsky, 1991; Taal, Rasker, Seydel & Wiegman, 1993a).

For example, Barlow *et al.*, (2002a) found in a longitudinal study of 60 RA out-patients that lower arthritis self-efficacy was associated with greater physical impairment, greater pain and fatigue, more depressed and anxious mood and less positive mood and acceptance 12 months later. Associations between other variables were as expected with positive correlations between physical health variables (HAQ, disease duration, pain and fatigue). Similarly, depressed mood was positively associated with HAQ, pain, and fatigue, and anxious mood was associated with HAQ and pain. Disease acceptance was negatively associated with HAQ, pain, fatigue, depressed and anxious mood and negative mood. Interestingly, partial correlations between physical health variables (HAQ, pain, fatigue) and psychological health variables (depressed and anxious mood, positive and negative affect

and acceptance), controlling for arthritis self-efficacy for pain and other symptoms were conducted. None of the correlations retained statistical significance, suggesting that the associations between physical and psychological health might be mediated by self-efficacy beliefs.

Research has also provided some information about how self-efficacy interacts with other factors influencing the self-regulation of RA. Schiaffino *et al.*, (1991) found that for people with RA reporting lower pain, self-efficacy was unrelated to depression one year later, but for those with higher pain, self-efficacy was associated with greater future depression. In this study self-efficacy also predicted the use of problem focused coping one year later, and Problem focused coping mediated the relationship between initial self-efficacy and later disability. Self-efficacy has also been shown to be associated with important self-management behaviours in RA including adherence to health recommendations (Taal *et al.*, 1993a) and adherence to prescribed medication (Brus, van de Laar, Taal, Rysker & Wiegman, 1999).

Shifren, Park, Bennett and Morrell (1999) found that self-efficacy mediated the relationship of intellectual functioning to positive and negative affect in people with RA. This study also found that older people with RA who had difficulty with cognitive tasks tended to report much lower self-efficacy, higher pain, and worse mental health outcomes. They suggest that intellectual functioning may affect mental health in RA in part because of its role in the cognitive representation of illness (Diefenbach & Leventhal, 1996). Being able to store access and use a wider variety of information about RA may provide individuals with the ability to maintain a broader view of RA and their ability to deal with it than can be managed by those with poorer intellectual functioning. There are multiple sources of information that individuals need to consider and integrate to make decisions regarding their physical and mental health (Diefenbach & Leventhal, 1996). Individuals with high intellectual functioning may be better at considering multiple sources of information and accessing both positive and negative dimensions associated with RA. Individuals with lower intellectual functioning may find it more difficult to contemplate multiple aspects of their situation and may focus only on negative

dimensions (Leventhal & Cameron, 1987; Swar *et al.*, 1999). Individuals who dwell on the negative dimensions are likely to develop increased symptoms (Smith, Christensen, Peck & Ward, 1994).

2.5.3(c) Helplessness

Parker *et al.*, (1989) have argued that in many respects, the Pain Control and Rational Thinking PCRT factor of the CSQ is similar to the construct of self-efficacy and, conversely, to another construct learned helplessness. Helplessness refers to the global state where individuals believe they lack viable ways to eliminate or alleviate sources of stress (DeVellis & Callahan, 1993). It has become increasingly clear that many people having arthritis develop a sense of helplessness when dealing with the daily challenges of their disease. This learned helplessness occurs when individuals come to expect that either negative outcomes will occur or positive outcomes will not occur, and simultaneously feel a lack of control over the occurrence of these outcomes (Smith, Peck, & Ward, 1990). Recent studies suggest that helplessness is particularly important in understanding the experience of people with RA, which is characterized by flares in disease activity that are often unpredictable and severe (Keefe *et al.*, 2002).

In research with people with arthritis, helplessness is typically assessed using the Arthritis Helplessness Index (AHI) developed by Nicassio, Wallston, Callahan, Herbert, and Pincus (1985). They administered the questionnaire to 219 people with RA, on two occasions separated by 1 year, with a number of other relevant variables. There was a significant negative correlation with Internal Health Locus of Control ($r = -0.62$), which would tend to suggest that these measures are sampling similar concepts and provides evidence for the validity of this instrument. In addition, they also found that initial high helplessness scores were associated with lower self-esteem, higher anxiety, depression and impairment in performing activities of daily living. In the longitudinal study, they found that changes in helplessness were associated only with difficulty with activities of daily living.

Further research has demonstrated that helplessness is associated with depression. Smith *et al.*, (1990) found in a cross-sectional study that helplessness accounted for the relationship between RA disease severity and depression. Although the result of this study is interesting, the direction of causal relationships is unclear. Smith *et al.*, (1994) conducted a 4- year follow- up of their original study and found that helplessness assessed at baseline was significantly related to higher levels of depressed mood 4 years later. These findings were obtained even after controlling for initial levels of depression.

Evidence of the relationship between helplessness and other self-regulatory processes in adaptation to RA comes from Smith and Wallston (1992). They conducted a longitudinal study of 157 people with RA. They examined the role of helplessness and passive coping in predicting changes in health outcomes that occurred over a 1- year period. Using sophisticated path analytic techniques, this study found clear evidence for the influence of helplessness. In fact, the pattern of findings suggested there might be a vicious cycle involving helplessness appraisals that lead to passive coping with pain and psychosocial impairment.

Schoenfeld-Smith *et al.*, (1996) conducted a longitudinal study to examine the role of helplessness in predicting disability in RA. A sample of 63 men with RA completed measures of disease activity, helplessness, pain, and psychological and physical function at three time points: baseline, 3 months, and 6 months. Path analysis revealed several interesting findings. First, disease activity did not have direct effects on either psychological or physical disability. Instead, disease activity was found to directly influence helplessness and pain. Second, both helplessness and pain were found to have direct effects on psychological disability and physical disability. Thus, the findings of this study suggest that helplessness and pain may mediate the relationship of disease activity to psychological and physical disability. Together, the results of longitudinal studies suggest that helplessness is important in understanding how persons adapt over time to their disease.

Nicassio *et al.*, (1993) conducted a longitudinal study that examined how helplessness was related to the outcome of a disease- modifying drug- treatment protocol. Participants were

50 people with RA, who completed a measure of helplessness before and after participating in a 3- month study of a disease- modifying drug. Cross- sectional analyses revealed that both before and after the drug trial, helplessness accounted for a highly significant amount of variance in measures of pain and disability. Most interestingly, helplessness assessed prior to the drug trial was a significant predictor of flares in disease activity after the drug trial. The results of these studies suggest a vicious circle in which greater disease activity leads to increased helplessness (Schoenfeld-Smith *et al.*, 1996) and greater helplessness predicts greater future disease activity (Nicassio *et al.*, 1993).

There is evidence that people with RA who have lower levels of formal education die at an earlier age (Pincus & Callahan, 1985). However, the mechanisms by which formal education is related to increased mortality are unknown. Callahan, Cordray, Wells and Pincus (1996) conducted a very important study in which they examined whether the relationship might exist because people with less formal education may feel helpless and unable to deal with their disease and its consequences. In this study, a cohort of 1,416 individuals diagnosed as having RA completed measures of helplessness, education, and disease activity at baseline and were then followed for 5 years. As expected, people with low formal education and higher levels of disease activity were found to have a significantly higher risk for early mortality. It is interesting, however, that when helplessness was entered into a model predicting mortality, education level was no longer found to be a significant predictor. These results suggest that helplessness may actually mediate the association of education level to mortality in people with RA.

Converging lines of evidence suggest that helplessness is an important construct in understanding pain, disability and emotional distress in RA. Helplessness has been found not only to relate to current and subsequent pain, disability and depression, but also to responsiveness to drug therapy. Most importantly, helplessness has been linked to early mortality, even after controlling for biomedical risk factors (Callahan *et al.*, 1996).

The research investigating cognitions and appraisals has arguably considered more divergent concepts than the work on coping strategies. However, many of these overlap

and as correlations between measures assessing helplessness and locus of control indicate, can be argued to be sampling similar concepts. It is clear from this research that illness related cognitions and appraisals and coping efforts are important in mediating the relationship between the disease and health outcomes. Although these results have not explicitly tested the SRM in RA, they provide substantial evidence which confirms some of the central tenets of this theory (Pimm & Weinman, 1998). Indeed, the model explicitly links illness representations, coping efforts and appraisal of coping with adaptation to illness. The research overwhelmingly confirms the role of these factors. Although the precise relationships between these concepts and different aspects of outcome are yet to be fully elucidated, they provide the basis for a preliminary understanding of the role of psychological factors in the course and outcome of RA.

2.5.4 Social context

People's attempts to understand and manage RA take place within a social context. Several aspects of the social context of RA have been investigated, for example social support, interpersonal and marital relationships, social comparisons and socio-economic status. Research has also considered how these factors may influence self-regulatory processes and health outcomes in RA.

2.5.4(a) Social support

Social support can be defined as the “processes by which interpersonal relationships promote well-being and protect people from health declines, particularly when they are facing stressful life circumstances” (Lanza & Revenson, 1993). There are different self-regulatory processes through which interpersonal relationships may influence health outcomes. For example eliciting social support is a coping strategy dependant on the availability of people in the environment who can provide support (Newman, 1998). Perceptions of the availability and quality of social support have also been shown to be important (Newman, 1998). Within Leventhal's SRM these might be viewed as part of the cognitive representation of the illness.

There is considerable evidence that social support is associated with health outcomes in RA. Ward and Leigh (1993) found, in a prospective study, that people with RA who were married had slower rates of progression of functional disability than unmarried people. They suggest that the emotional and practical support provided by the spouse acts to protect against increasing disability. In a study of people with RA who were in the early stages of their disease, Evers *et al.*, (1998) found that persons who had smaller social networks were much more likely to experience a decrease in mobility. A smaller social network has also been associated with depression in RA (Newman, Fitzpatrick, Lamb & Shipley, 1989).

However, most research has found that perceptions of the quality of social support are more important than the size of the social network or frequency of social interaction (Newman, 1998). People with RA who perceive themselves as having greater social support have greater self esteem (Fitzpatrick, Newman, Lamb & Shipley, 1988), life satisfaction (Smith *et al.*, 1991), better adjustment (Affleck, Pfeiffer, Tennen & Fifield, 1988a), less depression (Doeglas *et al.*, 1994; Fitzpatrick, Newman, Archer & Shipley, 1991), less fatigue (Riemsma *et al.*, 1998), cope better with arthritis (Mann & Zautra, 1990), and less work disability (Allaire, 1996).

One major area of controversy has been whether social support has a direct effect on health outcomes or whether it buffers the effect of stress on health outcomes in RA. There are two main hypotheses concerning the relationship between social support and health outcomes in RA. The first is known as the “main effect hypothesis” (Kraaimaat, Van Dam-Baggen & Biljsma, 1995a). This suggests that regardless of the illness severity or level of stress, social support is helpful. The alternative hypothesis is termed the “buffer effect hypothesis”, which proposes that social support moderates the relationship between stress and health outcomes. Specifically the argument is that it is only those who are under stress for whom social support is beneficial.

Brown, Wallston and Nicassio (1989b) conducted a longitudinal study investigating social support over time and its association with depression. Their study indicated that over time, social support appeared to be important, although it was the patients' perception of the degree of support rather than size of social network which was important. Their study provided evidence to support the main effect hypothesis rather than the buffer effect hypothesis over time. That is, regardless of the amount of stress, there remained a relationship between social support and depression.

The difficulty with this conclusion is that there are two potential confounds which make interpretation of the results complex. The first issue concerns the direction of any causal relationship. Patients' perceptions of social support are likely to be influenced by their mood. Indeed, in a cross-sectional design of the same study, there was an association between mood and negative perceptions of emotional support (Brown *et al.*, 1989b). Therefore, the relationship between social support and mood prospectively, could be an artefact of the well-documented relationship between mood and negative perceptions. In reality, the relationship is likely to be interactive. The second issue relates to the comparison of the buffering effect versus main effect hypothesis. Brown *et al.*, (1989b) argue that there is support only for the main effect hypothesis. However, one major difficulty in determining between these hypotheses in research on RA is the fact that RA in itself is a stressor, which produces illness related stresses with which people must cope in addition to the usual stressors of life. Therefore, it would seem difficult to argue coherently that a group of patients with RA could be identified who had either no stress or mild levels of stress.

Some support for the buffering effect hypothesis comes from a large study of people with arthritis. Penninx *et al.*, (1997) conducted a study of a community-based sample of 1,690 people, aged 55– 85 years, of whom 719 had no chronic disease, 612 had mild arthritis, and 359 had severe arthritis. This study provided evidence for the buffering effect of social support in that the results revealed that emotional support “mitigated the influence of arthritis on depressive symptoms” (Penninx *et al.*, 1997). Although research on social support has shown a relationship between social support and health, it remains unclear

whether social support has a direct effect on health outcomes or serves as a buffer against stress.

Although overall greater perceived social support is associated with positive health outcomes in RA the precise nature of the relationship is complex. Kraaimaat *et al.*, (1995a) investigated the role of both marital status and appraisals of social support in a cross-sectional design. They found that the marital status of female patients did affect mood, with divorced and widowed patients reporting higher levels of depression. However, they failed to find a difference in mood between people who were married and those who had never married. They also found significant differences between the divorced and widowed group and the other groups on ratings of social support, and the interaction between social support and psychological distress. That is, participants who were widowed and divorced saw their social network as significantly less supportive. Moreover, their negative perceptions had a greater effect on mood than the negative perceptions of married participants. Interestingly, Kraaimaat *et al.*, (1995a) found unexpectedly that a low level of functioning was associated with high levels of depression and anxiety but only for patients who were married. They interpret these results to indicate that high levels of pain and disability may have had negative effects on some relationships. Presumably becoming dependent upon someone as the condition deteriorates may have an impact on the relationship and subsequently mood. An alternative explanation may be that patients without a spouse have to continue with activities of daily living even when their joints are painful and they feel depressed. Those with (initially) supportive spouses may find spouses take over their responsibilities and hence they become less active. The loss of valued roles may lead to depression resulting in further reductions in functional ability. It seems likely that the relationships in these situations may indeed deteriorate, however, the direction of causality remains in question. This study highlights the complex nature of the relationships between social support and health outcomes in RA and indicates the need to understand more fully how perceptions of social support influence adaptation in RA.

2.5.4(b) Interpersonal and marital relationships

Some studies have examined how negative social interactions influence emotional well-being in RA. Manne and Zautra (1990) report a study of marital interactions and adjustment in 103 women with RA and their husbands. They found that in couples in which the husband made more critical remarks about the patient, the patient had higher levels of pain and disability, and the husband showed poorer psychological adjustment. In couples in which the husband was supportive, the patient showed better psychological adjustment. It is interesting that negative interactions with a spouse played a more significant role in explaining adjustment of the patient than supportive interactions.

In a separate report from this study, Mann and Zautra (1989) found that social support was associated with coping behaviours and psychological adjustment. Women with critical spouses engaged in more maladaptive coping strategies (e.g. wishful thinking) and had poorer psychological adjustment. Women who perceived spouses as supportive used more adaptive coping strategies (e.g. information seeking and cognitive restructuring). They suggest that the spouse may influence psychological adjustment indirectly by influencing the person with RA's selection of maladaptive or adaptive coping efforts.

Kraaimaat *et al.*, (1995b) examined how spouses' reactions were related to psychological distress in men and women having RA. When spouses responded in a critical fashion, men having RA reported significantly higher levels of anxiety, and women having RA reported significantly higher levels of anxiety and depression. These findings suggest that negative aspects of social interactions may be related in important ways to adjustment to arthritis.

Some studies have investigated different types of social support and have found that not all types of social support were associated with improved health status or emotional well-being. Taal *et al.*, (1993a) found emotional support was not positively related to health status but practical support was. In a study of 54 people having RA, Doeglas *et al.*,

(1994) found that persons who received higher daily emotional support experienced higher levels of psychological well-being. However, problem-oriented emotional support was negatively related to some aspects of psychological well-being. Persons who had a higher degree of social companionship were also less depressed. Further research is needed to clarify which people are likely to benefit from what type of social support, provided by whom and in what circumstances. Future research needs to focus more on the different dimensions of social support (e. g., emotional support and instrumental support), as these might show different relationships to pain, disability, and other health outcomes. It is also important to identify the individuals who are most likely to benefit from social support.

2.5.4(c) Social comparisons

A third process, by which social factors might influence self-regulation of RA, is social comparisons. As part of the process of appraisal of the illness and coping efforts people make comparisons with others to evaluate their relative position. Social comparisons may be particularly important when it is not possible to change the situation (Wills, 1987).

Affleck, Tennen, Pfeiffer, Fifield and Rowe (1987c) found in 90 people with chronic RA that they made more downward comparisons. That is, they compare themselves with another person with RA with poorer health status. They proposed that this enables them to view their health status as better and preserve their emotional well-being. The results of this and later studies confirmed that people with RA who made downward social comparisons had better emotional well-being (Affleck, Tennen, Pfeiffer, & Fifield, 1988b; Devellis *et al.*, 1990).

2.5.4(d) Socio-economic status

As discussed earlier, social factors (e.g. educational attainment and income) are related to mortality and morbidity in RA (Callahan *et al.*, 1996; Pincus & Callahan, 1985). In RA, those who have lower levels of formal education have been found to have significantly higher mortality rates (Callahan *et al.*, 1996; Pincus & Callahan, 1985). This relationship appears to be independent of age, medication use, functional status, or disease duration (Pincus & Callahan, 1985).

Socio-economic status has been related to health outcomes and self-regulatory processes in RA. Studies have shown that low socio-economic status is related to higher functional disability, depressive symptoms, and maladaptive coping styles (Berkanovic *et al.*, 1996; Downe-Wamboldt & Melanson, 1995). One study compared 141 people with RA living in an affluent area with 106 people with RA living in a less-affluent area of the same city (Brekke, Hjortdahl, Thelle, & Kvien, 1999). Participants were administered measures to assess the disease process, joint damage, health status, health-related quality of life, and arthritis self-efficacy. The results indicated that both groups had similar levels of joint damage and disease severity. However, those who lived in the less-affluent area reported significantly poorer health status and lower self-efficacy.

The pathways linking low socio-economic status and increased mortality and morbidity in RA are unclear. One possibility is that people with low formal education have lifestyles that predispose them to arthritis and other diseases. Another possible explanation, discussed earlier, is that the relationship between educational level and mortality is mediated by learned helplessness (Callahan *et al.*, 1996). Third, it is possible that limited access to health care may account for higher morbidity among low socio-economic status groups.

2.6 Review of research using the self-regulatory model in RA

Pimm and Weinman (1998) have adapted Leventhal's self-regulatory model in an attempt to explain how the experiences of people with RA affect their responses and their adaptation to illness. They propose that prior to diagnosis, people have different representations about illness based on their personal experiences. Their experience of RA then gives rise to two levels of response, the cognitive representation of the health threat and the representation of the emotion. These representations are thought to underlie a person's choice of strategy for coping both with symptoms of the illness and ensuing emotions. These efforts to cope with the illness and its consequences are in turn appraised by the individual in a process of adaptation. These illness representations are hypothesised to guide coping efforts and subsequently affect disease outcome. As such, they should be amenable to cognitive behaviour therapy with the aim of facilitating the development of adaptive representations and coping efforts.

2.6.1 The development of illness representations

As discussed above a variety of factors are hypothesized to contribute to the development of illness representations. Although most research has examined the illness representations of people with established RA some studies have investigated people without RA or at earlier points in the development of RA.

Research guided by the SRM has been facilitated by the development of questionnaires, such as the Implicit Models of Illness Questionnaire (IMIQ; Turk, Rudy & Salovey, 1986) and the Illness Perceptions Questionnaire (IPQ; Weinman, Petrie, Moss-Morris & Horne, 1996) specifically designed to measure the cognitive representations of illness proposed in Leventhal's SRM.

According to self-regulatory theory, even people without illness have various representations about specific illnesses, which are based on their indirect experiences, which may include media and cultural images. These representations, which exist prior to

developing an illness, are thought to change in the light of the illness characteristics and the personal experience of illness to create the illness representation.

Schiaffino and Cau (1995) examined the difference between student and patient representations of three illnesses, RA, multiple sclerosis (MS) and human immunodeficiency virus (HIV), using the IMIQ. Their results indicated that student representations of illnesses were very different from patient representations. With regard to RA, students viewed the illness as more curable and less variable than patients did. Students also felt that there was a higher level of personal controllability for the illness than patients believed. Interestingly, both groups saw RA as equally serious. This research supports the view that even people without RA hold implicit models of the illness. Presumably, an individual's previous conceptualisations of a particular illness are likely to affect their initial responses to the illness and the further development of their illness representation.

Schiaffino and Cau's (1995) results would indicate that pre-existing representations of RA are likely to be consistently different to the representations that develop as a result of living with chronic R.A. According to these results, it seems likely that patients use their developing knowledge of the illness and their personal experience of symptoms to change their illness representation following diagnosis. Hence, illness severity and other illness characteristics are likely to influence and change illness representations during the course of adjustment. Recent research has investigated how these processes of change occur and what factors may be important in the development of adaptive representations of illness in RA.

There is evidence that implicit models of the illness shape people's responses at the pre-diagnostic and early phases of RA. Donovan (1991) interviewed people referred to rheumatology clinics prior to their first consultation and diagnosis with RA and found that they had well developed lay models of their illness which affected their response to the information they received from health professionals. Sakalys (1997) interviewed 50 women in the pre-diagnostic stage of RA. This study revealed a high incidence of symptom normalization, self-treatment, symptom comparison, and prolonged time to diagnosis with multiple misdiagnoses and physicians consulted. Most participants reported invalidation of

initial symptoms, multifaceted emotional distress, and relief upon accurate diagnosis.

Significant associations were found between:

- (a) illness related symptom attributions and fewer physicians consulted and less invalidation;
- (b) life stress events and fewer physicians consulted and shorter time to diagnosis;
- (c) remissions and time to diagnosis, number of physicians consulted, and number of misdiagnoses.

This study illustrates the important role that self-regulatory processes play in people's early attempts to make sense of RA.

The early years of RA may be a particularly important phase in the development of illness representations as new information is accommodated into pre-existing representations. In addition, these early illness representations may play an important role in determining later adaptation. For example, Sharpe, Sensky and Allard (2001a) investigated a small group of 22 people with sero-positive RA of less than two years duration longitudinally. They found that baseline levels of depression, pain, disability and perceiving RA as having more serious consequences (assessed on the IPQ) predicted higher levels of depression up to 21 months later.

2.6.2 Self-regulatory processes in chronic RA

It seems likely that people experiencing similar challenges associated with a particular rheumatic disease may develop some common illness representations. Hampson *et al.*, (1994) report the shared beliefs of people with OA assessed using a 60 item structured interview (the Personal Models of OA interview) developed to assess multiple dimensions of the illness representation. A majority (75%) viewed OA as fairly/moderately serious, with unpredictable symptoms (81% reported pain varied daily); as chronic (93% expected to have OA permanently); incurable (79% believe a cure is unlikely). In this sample 94% believed it is important to prevent OA getting worse and that it can be managed using medical treatments (95% believe treatment makes them feel better and 88% felt no aspect of their treatment made them feel worse).

Scharloo *et al.*, (1999) assessed 71 people with RA using the IPQ (Weinman *et al.*, 1996). With respect to the Identity construct they found that in addition to pain people viewed a wide range of other symptoms (e.g. fatigue, sleep difficulties, stiff/sore joints, and loss of strength) as salient features of their illness. On average people with established RA perceive RA as having multiple and frequent symptoms, adverse consequences, a chronic time line, that they have some control over their disease and many believed stress was an important causal factor in the development of their RA.

A number of studies have investigated the extent to which differences in illness representations explain emotional and behavioural responses and health outcomes in RA. In a cross-sectional study, Scharloo *et al.*, (1998), investigated the relationship between cognitive representations of illness, coping and functional outcomes. This study included a group of 84 people with RA, as well as people with chronic obstructive lung disease and psoriasis. The results indicated that a strong illness identity, believing the illness was likely to be chronic, and have serious consequences and the use of passive coping behaviours were associated with poorer functional outcomes (assessed with illness specific measures and the general role and social functioning scales of the Medical Outcomes Study SF-20). In contrast, seeking social support and beliefs that the illness was controllable were associated with better outcome.

The relationship between cognitive representations of illness, coping and depression has also been investigated (Murphy, Dickens, Creed, & Bernstein, 1999). Using a cross-sectional design 62 people with RA completed the Hospital Anxiety and Depression Scale, IPQ, London Coping with RA Questionnaire, the HAQ and a clinical assessment of their physical state. Depressed patients were more disabled than the non-depressed, had a more negative view of their illness, and used more negative coping strategies. However, when disability was controlled for, the relationship between depression and negative coping strategies was no longer significant. Interestingly the association between higher levels of depression and both perceiving the consequences of RA to be more serious and viewing RA as less controllable remained even when disability was

controlled for. This provides evidence that greater perceived consequences do not simply reflect the fact that RA is actually more serious. This study indicates the strong association between illness representations and depression. However as this study is cross-sectional, it is impossible to make causal statements about these relationships. It is possible that people view their illness more negatively because they are depressed, or people may become depressed because they view their illness so seriously and feel they have no control over it. Depression is disabling and is associated with a more negative perception of illness and other aspects of life (Murphy *et al.*, 1999) However, there is evidence from longitudinal work suggesting that negative cognitive processes precede depression in RA patients (Smith *et al.*, 1994). Murphy *et al.*, (1999) suggest that longitudinal intervention studies in which illness representations are modified may help to clarify the complex bi-directional relationships between pain, disability and depression in RA.

While the results of these studies in chronic RA confirm the association of the relevant representations with health outcomes, they shed little light on the process of adaptation across the course of the disease. The self-regulatory model proposes that those with maladaptive illness representations are likely to cope more negatively and subsequently develop poorer outcomes. However the direction of causal relationships is unclear. An alternative model might suggest that those with the poorest prognosis learn, on the basis of their negative experiences, to have illness representations that are more negative than those with more positive experiences of illness. If the beliefs are more accurate then a poorer outcome would be expected. The data from cross-sectional work in chronic illness, such as that by Scharloo *et al.*, (1998) and Murphy *et al.*, (1999) cannot differentiate between these hypotheses. Indeed, models suggesting a linear relationship in either direction are likely to be mechanistic and are unlikely to account for the complex relationships that mediate illness representations and health outcomes.

To understand the complex dynamic relationships between self-regulatory processes and health outcomes, studies assessing a range of self-regulatory processes and health outcomes over time are needed. Scharloo *et al.*, (1999) extended their earlier work conducting a 1-year follow-up of 71 people with established RA. They investigated the relationship between illness perceptions and coping at the beginning of the study and health outcomes one year later. Using multiple regression analysis they controlled for baseline levels of the health outcomes, illness duration and disease severity at the time of the second assessment. They found that illness perceptions and coping strategies explained a significant amount of variance in health outcomes 1 year later. A stronger illness identity (perceiving their RA as having a greater range and frequency of symptoms) was associated with more pain, more tiredness, and more depression one year later. Perceiving RA as having more serious consequences was associated with more visits to the outpatient clinic, more tiredness, and higher anxiety scores. A chronic time line, belief that the illness will last a long time was associated with higher anxiety scores. Perceiving RA as less controllable was associated with more hospital admissions. A number of coping strategies were also associated with health outcomes. Less expression of emotion was associated with more hospital admissions. High scores on coping involving fostering reassuring thoughts were associated with more functional disability. More passive coping was associated with more functional disability and higher anxiety scores. More avoidant coping was associated with more tiredness. Baseline scores for the health outcomes usually accounted for most of the variance in the health outcomes at one year. As there was significantly less variance to explain, the amount of variance explained by illness perceptions and coping strategies was relatively small, varying between 2-20%. However, this is still impressive as illness perceptions and coping strategies were explaining variance in health outcomes 1 year later and frequently explained more variance than the illness duration or concurrent disease severity.

Sharpe *et al.*, (2001a) have observed that, although prospective studies of chronic RA do reveal how established illness representations influence future health outcomes, they tell us little about the processes by which people develop illness representations. They also note that prospective studies assessing relationships between factors over long time periods are

likely to miss the subtle interaction between self-regulatory processes that occur over relatively short periods of time. However Leventhal *et al.*, (1992a) suggest that at different phases of an illness different self-regulatory processes are likely to be salient. Therefore, when attempting to explain how illness representations develop, studies of early RA are needed. However when attempting to understand how people with chronic RA perceive and respond to RA, and the implications of these for their future health, studies of chronic RA, are needed.

It might be argued that, in established RA, illness representations may change little over time, which limits the extent to which the dynamic nature of self-regulatory processes may be explored. An alternative methodology for investigating the dynamic relationships between self-regulatory processes and health outcomes may be to experimentally manipulate self-regulatory variables in longitudinal intervention studies. Interestingly Sharpe *et al.*, (In press) have used this methodology to study 23 people with sero-positive RA of less than two years duration participating in a CBT intervention (see Chapter 3).

A number of key points emerge from this review. First in RA cognitive representations of illness and coping procedures are associated with health outcomes in both cross-sectional and longitudinal studies. The most consistent findings are that a stronger illness identity, perceiving RA to be chronic, difficult to control and to have serious consequences are associated with poorer physical and psychological adaptation and greater use of health services. Second no studies have explicitly investigated emotional representations or appraisal processes. Third the complex dynamic relationships between self-regulatory processes and health outcomes are difficult to disentangle. Longitudinal and intervention designs assessing self-regulatory processes and health outcomes at multiple time points may elucidate these relationships. Studies at one point in the development of an illness may provide little information about the role of self-regulatory processes at different phases of the illness.

2.7 Summary

There are considerable individual differences in the levels of pain, disability and emotional distress experienced by people with RA. Psychosocial factors usually account for a greater proportion of the variance in pain, disability and depression than objective measures of disease activity (Young, 1992).

With no cure for RA, and with medical treatments only being partially effective, it is important to explore the self-regulatory processes that mediate between the disease, pain, disability, and psychological adjustment. Understanding how some people successfully make sense of and manage RA, minimizing pain and disability and optimizing psychological adjustment, may help in the development of interventions to assist people who are managing relatively poorly.

There is an extensive research literature that has explored the role of psychosocial processes in adaptation to RA. This review indicates that illness-related cognitions, coping behaviours, appraisals of coping efficacy and social factors influence physical and psychological outcomes in RA. There is also evidence that these factors interact in a complex and dynamic manner.

Leventhal's SRM has been found to be a useful framework for understanding adaptation to acute and chronic illness (Petrie & Weinman, 1997), however, only a few studies in RA have been guided by this model. In early and chronic RA, cognitive representations of illness and coping procedures have been associated with health outcomes in both cross-sectional (Murphy *et al.*, 1999; Scharloo *et al.*, 1998) and longitudinal studies (Scharloo *et al.*, 1999; Sharpe, 1999). The most consistent findings are that poorer physical and psychological adaptation and greater use of health services are associated with:

1. Perceiving RA as having a stronger illness Identity, more serious consequences and to be more difficult to control;

2. Greater use of passive and less use of active coping strategies. Although these findings are interesting, there have only been three studies and these have significant methodological weaknesses (e.g. small sample size, cross-sectional design or only examining a limited number of illness representations and coping procedures).

The complex dynamic relationships between self-regulatory processes and health outcomes are difficult to disentangle. Longitudinal and experimental designs, assessing and manipulating self-regulatory processes, and evaluating their impact on health outcomes, at multiple time points may elucidate these relationships.

Chapter 3: Psycho-educational interventions for people with RA

3.1 Introduction

There is increasing acceptance that Psycho-educational interventions have potential to make a significant contribution to the successful management of RA (Astin, Beckner, Soeken, Hochberg & Berman, 2002; Huyser & Parker, 2002; Keefe *et al.*, 2002). This chapter starts with a discussion of the rationale, definition and aims of psycho-educational interventions for people with RA. The types of psycho-educational intervention methods that have been investigated are then described. Next the evidence for the efficacy of psycho-educational interventions is reviewed, followed by a discussion of studies that have explored mechanisms of change in psycho-educational interventions. Finally, the literature is summarized and areas for further research are identified.

3.2 Rationale for psycho-educational interventions

There are a number of reasons for the increased interest in the development and evaluation of psycho-educational interventions for people with RA. First, the traditional approach to patients with RA has been pharmacological, often in combination with physical therapy, and sometimes with surgery. Although useful, these interventions are only partially effective in relieving symptoms (20-50% improvement (Hirano *et al.*, 1994) and preventing disease progression. Thus many people are left with distressing symptoms, disabilities, and uncertainty about the future course of their illness.

Second, there is increasing recognition that treatment for RA needs to be multifaceted, directed not only at controlling the disease and its physical consequences but also at the social and psychological functioning of the patient (Keefe *et al.*, 2002) However, currently rheumatological care frequently fails to address psychosocial issues such as psychological well being, social or occupational function (Hawley, 1995). For example,

as part of a larger study Barlow *et al.*, (2002a) interviewed people with RA, attending an UK outpatient rheumatology clinic, about the source and content of information they had received. The outpatient clinic was the main source of education about the disease and its treatment. One exception concerned information about side effects, which appeared to be obtained largely from pharmacists or prescription drug leaflets. Education about self-management was received mainly from physiotherapists and occupational therapists, and focused primarily on exercise, use of aids and footwear. Patients could not recall receiving education, information or advice about pain-management, coping with flares, psychosocial issues (e.g. emotions, work and relationships).

Therefore for many people with RA pain, fatigue, disability, psychological distress, and poor quality of life persist in spite of treatment (Hawley, 1995). To optimise the quality of life for people with RA there is a major need for interventions that address residual distressing symptoms, physical function, emotional well-being and restricted social roles.

Third, treatment of RA makes major demands on the person. It necessitates frequent consultations with health professionals, complex and aversive medication regimes, the prospect of major surgery and modification of lifestyle and the adoption of a wide range of self-management strategies. Responsibility for the daily management of arthritis gradually shifts from the health care professional to the individual. Successful self-management of chronic conditions requires sufficient knowledge of the condition and its treatment, performance of condition management activities and application of the necessary skills to maintain adequate psychosocial functioning.

Although there is no gold standard definition of self-management it is interesting that recent definitions have emphasized the importance of self-regulatory processes.

"Self-management refers to the individual's ability to manage the symptoms, treatment, physical and psychosocial consequences and life style changes inherent in living with a chronic condition. Efficacious self-management encompasses ability to monitor one's condition and to affect the cognitive, behavioural and

emotional responses necessary to maintain a satisfactory quality of life. Thus, a dynamic and continuous process of self-regulation is established."

Barlow (2001b).

Self-management of RA involves performance of a wide range of therapeutic behaviours (e.g. medication taking, exercise, splint wearing and clinic attendance). These therapies are often aversive and the benefits are seldom evident in the short-term, as they are needed to delay further progression of the disease and prevent disability. Given the complexity, aversiveness and long-term nature of therapies for RA it is not surprising that adherence is often poor, reducing the benefit of the therapies and leading to increased health-care costs. Bradley (1989) reviewed treatment adherence in RA and found that rates of adherence varied from 33-78% for medication and 33-66% for exercise. As discussed in Chapter 2 psychosocial processes (e.g. self-efficacy; Brus *et al.*, 1999; Taal *et al.*, 1993a) are thought to be important determinants of adherence.

Fourth, another important development is the shift away from paternalistic models of health care that sited the patient in the role of passive recipient. Many patients now expect more active involvement in their health care. This is consistent with the realities of chronic disease, discussed above, whereby responsibility for day-to-day disease management gradually shifts from health care professionals to the individual. Indeed, the UK initiatives such as NHS Direct and the Expert Patients Task Force are based on the notion of patients as experts able to access information relevant to their health care needs and to carry out the self-management tasks needed for their condition at a given point in time (Department of Health, 2001). Support for this approach comes from the research described in Chapter 2. Active participation in treatment decisions and procedures helps people combat helplessness, develop a sense of self-efficacy, and enhance emotional well-being. People seek to understand their illness developing a working model of what the illness is, its effects, why it has happened, how long it will last and whether it can be cured or controlled. There is evidence that these personal models influence emotional responses to the disease coping strategies, and health outcomes (Scharloo *et al.*, 1999). Therefore interventions aimed at assisting people to develop adaptive models of their disease and

encourage active participation and self-management may produce significant health outcome benefits.

Finally, an important reason for increased interest in psycho-educational interventions is the shift in the demographic profile. Life expectancy is increasing and more people are living with chronic conditions. The burden of meeting the needs of this growing number of people will fall upon already over-stretched health care services, that are struggling to cope with the demands of acute care, let alone the needs of those with long-term health conditions. Treatment of RA makes major demands on health service resources; it is expensive, non-renewable and often associated with side effects. Psycho-educational interventions may enable more efficient use of limited resources, by reducing the need for medical consultations, provision of equipment, and use of analgesics. It is also possible that psycho-educational interventions may reduce the socio-economic burden of RA on the individual and society.

Given these reasons it is not surprising that the potential of psycho-educational interventions to enhance self-management of chronic illnesses is the subject of increased attention and was mentioned in recent White papers in the UK (Department of Health, 1999). Self-management may be one means of bridging the gap between patients' needs and the capacity of health and social care services to meet those needs

3.3 Definition and aims of psycho-educational intervention

People with RA require sufficient knowledge and understanding to make decisions regarding treatment options and lifestyle changes, but they also need the necessary skills and confidence to manage RA in everyday settings. Psycho-educational interventions can be operationalized in accordance with the definition of health education, proposed by Tones (1997), as intentional activities designed to achieve disease-related learning and that may lead to changes in knowledge, understanding, beliefs, skill acquisition or behaviour change. Psycho-educational interventions may encompass information-based

materials, self-management programmes, cognitive-behavioural or other forms of psychosocial intervention.

There are four main aims of psycho-educational interventions for RA (Tucker & Kirwan, 1991). First, improving or maintaining optimal health status. The most important areas of health status include: pain and other symptoms such as fatigue, physical function, emotional well-being, social interaction and occupation, and disease activity. Second, to assist people to change health related cognitions and behaviours that have been found to be important in mediating between the disease and its outcome. Third, to improve the use of health service resources. Fourth, to improve the acceptability or satisfaction with treatment.

3.4 Psycho-educational intervention methods

Psycho-educational intervention is an umbrella term that encompasses both educational and psychological interventions. Many psycho-educational interventions combine an educational intervention and psychological intervention. Interventions may be delivered to an individual or group and in clinical settings or in the community. In addition, support from family, professionals, and other people with rheumatic disease may be employed.

Many different psycho-educational interventions for rheumatic disease have been developed and evaluated in controlled research studies. These can be broadly categorized into four types (DeVellis & Blalock, 1993):

1. Education or structured provision of information (e.g. leaflets, books, Didactic classes and computer assisted learning);
2. Self-management programmes. The best known is the Arthritis Self-Management Programme (ASMP) developed by Lorig and her colleagues at Stanford University (Lorig, Lubeck, Kraines, Seleznick & Holman, 1985);

3. Cognitive behaviour therapy (e.g. relaxation, biofeedback and pain management);
4. Other psychosocial interventions (e.g. psychotherapy, social support and family therapy).

Psycho-educational interventions must be carefully planned to be effective (Burckhardt, 1994; Daltroy & Liang, 1993; Green & Kreuter, 1991). This begins with a thorough analysis of the needs of the patients, of the health problems they experience, of the behaviour associated with their needs, and of the determinants of such behaviour (Taal, Rasker & Wiegman, 1995). Psycho-educational interventions are designed and implemented on the basis of this analysis.

Hirano *et al.*, (1994) reviewed 20 needs assessment and process evaluation studies of people with rheumatic disease. The most commonly reported problems and needs of people with arthritis included pain, fatigue, uncertainty about future, depression and lifestyle change/adjustment. Rheumatologists were the most valued source of information. Other sources included books/pamphlets, nurse practitioners and telephone services. Interestingly these studies found differences between patients and doctors perceptions of their needs especially in the area of compliance to treatment recommendations.

Even people who have had RA for a long time, who have had regular contact with health professionals, perceive their need for information about how to manage RA to be as great as those who are more recently diagnosed. Barlow, Cullen and Rowe (1999a) found that for people with RA, attending an UK rheumatology outpatient clinic, the need for information, education and advice was similar, regardless of disease duration or severity. Participants reported that they needed help in the following areas: understanding RA and its treatment; management of pain and fatigue; coping with flares; aids and adaptations; impact on work, family, relationships, and emotions. In addition many people expressed a wish to know more about their prognosis and how they might influence this.

Barlow *et al.*, (2002a) investigated preferences for methods of delivery of psycho-educational interventions of people with RA attending a rheumatology outpatient clinic. Preferred methods for delivery depended on the topic. They preferred education about the disease and its treatment to be delivered on a one-to-one basis by health professionals. Similarly, emotional issues were believed to be best dealt with one-to-one although this could be with a similar other (i.e. a patient). Group interventions were the preferred format for self-management, exercise and relationship issues, whereas videos were thought to be useful for demonstrating use of aids and how other families cope. None of the participants welcomed computer-based interventions.

These studies provide a foundation for the development of psycho-educational interventions. They have also led to the development of criteria for psycho-educational interventions for rheumatic disease (Taal *et al.*, 1995).

Self-management and CBT interventions have been more thoroughly investigated than other psycho-educational interventions (Hawley, 1995; Taal *et al.*, 1995). Hawley (1995) distinguishes between self-management and cognitive behavioural interventions as follows:

"Self-Management / Self-Help programmes

Self-Management programmes provide a combination of

1. Disease-related information and
2. Assistance in learning and adapting new activities and skills.

Information: pathology, medications and techniques e.g. joint protection.

Activities: exercise, relaxation and energy saving techniques.

Skills: assertiveness with professionals and family, time management, problem solving. Emphasis is on changing behaviours by presenting information and using group interaction and mutual support. Recent programmes emphasize improving self-efficacy. Programmes may be led by laypersons or professionals.

Cognitive behavioural therapy

Emphasis is on learning new techniques for controlling or managing pain. CBT is based on the premise that cognitive and behavioural changes can help one control pain. Generally there are three phases:

1. Educational (e.g. simple explanation of pain theories and the interrelationship of the emotional, cognitive, physical and behavioural components of pain),
2. Learning new skills (e.g. relaxation, diversion, cognitive restructuring) and
3. Maintenance (e.g. transfer new skills to everyday life).

(Parker, Iverson, Smarr & Stucky-Ropp, 1993). Generally led by psychologists or specially trained professionals."

(Hawley, 1995).

However the distinction between self-management and CBT interventions is not as clear-cut as these definitions suggest. Self-management and cognitive-behavioural interventions did have different theoretical origins. Self-Management programmes were originally developed using models of health education. However, self-management interventions have increasingly incorporated an understanding of psychological theory and the use of cognitive-behavioural strategies (Lorig & Gonzalez, 1992; O'Leary, Shoor, Lorig & Holman, 1988). Cognitive behavioural interventions were originally based on models such as the Gate Control Theory (Melzack & Wall, 1996). As a result, the scope of early CBT interventions tended to be narrower than self-management programmes. However more comprehensive cognitive-behavioural interventions have been developed which have expanded, beyond the focus on pain management, to address the wider physical and psycho-social consequences of RA (Parker *et al.*, 1995).

Studies of self-management interventions have most often investigated mixed diagnostic groups, including people with several different types of arthritis. Participants are usually recruited from community populations, using methods such as newspaper advertisements. However, the ASMP has also been evaluated in single diagnostic groups, recruited from hospital clinics (e.g. RA; Taal *et al.*, 1993b). Cognitive behavioural interventions have been most widely applied with people with RA attending hospital clinics. However, the

value of CBT for people with other forms of arthritis has also been investigated in a few studies (e.g. OA; Keefe *et al.*, 1990a; Keefe *et al.*, 1990b).

Self-management programmes are usually led by trained lay people whereas CBT interventions tend to be led by psychologists or other specially trained health professionals. However, in some studies self-management programmes have been delivered by health professionals (Cohen, Sauter, DeVellis, & DeVellis 1986; Lorig *et al.*, 1986). Overall, although broad historical distinctions can be drawn between self-management and CBT interventions, in current programmes there is a degree of convergence in theoretical basis, content and methods of delivery. However, few studies have evaluated self-management programmes, led by health professionals, for people with RA recruited from hospital outpatient populations.

3.5 Efficacy of psycho-educational interventions

There have been over 100 peer-reviewed published studies of psycho-educational interventions for rheumatic disease (Lorig, 1995). The trials have varied in both the content of the programme and the populations from which their samples have been drawn. This section begins with a review of the efficacy of each of the four main psycho-educational interventions with illustrative studies followed by a discussion of reviews and meta-analyses of this literature.

3.5.1 Educational interventions

The value of providing structured information about rheumatic disease has been thoroughly investigated. The majority of studies show that education programmes are successful in increasing knowledge about the illness and some studies have found increased use of self-management behaviours, such as exercise and joint protection (Lorig, Konkol & Gonzalez, 1987). Although a few studies have shown that provision of information leads to improvements in health status (e.g. pain, disability and depression) most have failed to find such benefits (Lorig *et al.*, 1987). Some studies have even found

the reverse relationship. For example, Parker *et al.*, (1984) presented a 7-hour education programme to people with chronic RA. Although the intervention group showed increases in knowledge they also reported more pain and disability than the control group. Similarly, Brus, Taal, van de Laar, Rasker & Wiegman (1997) found that while people with RA with low disease activity experienced a reduction in anxiety following an education package, the reverse was true for people with severe disease. These findings are confirmed by Superio-Cabuslay, Ward and Lorig, (1996) who report a meta-analysis which found that the effect sizes for educational interventions for OA and RA were smaller than for psycho-behavioural interventions (Pain 0.07 0.18; disability -0.19 0.05; tender joint count -0.12 0.43). Overall, while many people with rheumatic disease are not adequately informed about their conditions (Daltroy, 1993), most evidence also suggests that merely providing information has limited value (DeVellis & Blalock, 1993). In the light of the limited success of educational programmes for RA, various self-management, cognitive behavioural and other psychosocial interventions have also been developed.

3.5.2 Self-management interventions

The literature on self-management interventions in arthritis is dominated by the Arthritis Self-Management Programme (ASMP). The ASMP was designed as a community-based, group approach for people with mild-moderate arthritis, led by lay tutors and accompanied by a manual for participants and tutors (Lorig & Holman, 1993). It is set within the framework of Self-Efficacy Theory (Bandura, 1977). A central tenet of the ASMP is to increase participants' perceptions of arthritis self-efficacy defined as perceived ability to control, or manage, various aspects of arthritis.

Randomised controlled trials conducted in North America have shown the ASMP to be effective in terms of increasing knowledge about arthritis, performance of self-management behaviours, perceptions of self-efficacy, decreasing pain, depressed mood and fewer visits to doctors at 4-month follow-up (Lorig & Holman, 1993). Improvements remained evident at 20 months (Lorig & Holman, 1989) and at 4 years (Lorig, Mazonson & Holman, 1993). The long-term effects of the ASMP do not appear to be enhanced by

reinforcement through bi-monthly newsletters or attending a second ASMP course designed to reinforce the principles of self-management (Lorig & Holman, 1989)

The reduction in frequency of medical consultations, found following self-management interventions, has been estimated to make significant cost savings for OA and RA. Lorig *et al.*, (1993). conducted a 4 year follow-up study of 401 participants of the ASMP and found a 40% decrease in doctor visits over baseline, a mean decline in slightly more than two visits per year. For RA patients (n=75) the decrease in visits was 49% (mean decrease of 5.1 visits per year) with an estimated cost saving of \$648. Doctor visits for OA patients (n=263) decreased 39% with a cost saving of \$189 (Lorig *et al.*, 1993).

The ASMP is usually delivered as a group programme. A recent review of self-management for chronic illness concluded that group approaches appear to be as effective as individualised approaches (Barlow, Wright, Sheasby, Turner & Hainsworth, 2002b). However, one major issue with individualised approaches involving one-to-one contact with health professionals is the cost of the intervention. Moreover, it was found that simply sharing experiences of arthritis with others in a safe and non-threatening environment was a hidden benefit of ASMP attendance, that reduced feelings of isolation (Barlow, Cullen, Davis & Williams, 1997a).

There appears to be little difference in the effectiveness of the ASMP when delivered by trained lay leaders with arthritis or health professionals (Cohen *et al.*, 1986; Lorig *et al.*, 1986). A RCT has even shown that a mail delivered Arthritis Self-Management Programme along with individualised, computer-generated advice produced similar improvements to the group programme (Fries, Carey & McShane, 1997). At 6 months, participants had decreased pain, improved joint count, increased self-efficacy, increased exercise and made fewer visits to doctors.

Although the ASMP was originally developed in the USA it is now used in many countries (e.g. Canada, Australia, New Zealand, and European countries including the UK (Hawley, 1995). Studies of modified versions of the ASMP delivered in Australia

and the Netherlands reported similar improvement, but failed to find significant reductions in pain after 12 (Lindroth, Bauman, Barnes, McCredie & Brooks, 1989) and 14 (Taal *et al.*, 1993b) months, respectively. In the UK, the ASMP has been evaluated in a range of samples and delivery settings, focusing on change over time (Barlow, Williams & Wright, 1997b; Barlow, Turner & Wright, 1998a; Barlow, Turner & Wright, 1998b; Barlow, Williams & Wright, 1999b; Barlow, Shaw & Harrison, 1999c)

A recent RCT of the ASMP in the UK demonstrates that this programme can transcend national Boundaries (Barlow, Turner & Wright, 2000). Participants were 544 people with arthritis recruited from the community. They were randomly allocated to either ASMP (Intervention Group) or a 4-month waiting list Control Group. The Intervention Group completed a 12-month follow-up. At 4-month follow-up, the ASMP had a significant effect on arthritis self-efficacy for other symptoms and pain subscales. Performance of a range of health behaviours (cognitive symptom management, communication with doctors, dietary habit, exercise and relaxation) was significantly greater among the Intervention Group. The Intervention Group were significantly less depressed and had greater positive mood. There were no changes in physical functioning, pain and GP visits at 4-month follow up. A similar pattern of findings was found at 12-month follow-up for the Intervention Group. However, a significant reduction in pain and visits to GPs were also found. Apart from a small improvement on physical functioning for participants in the intervention group with osteoarthritis, at 12 months, all effects were independent of the type of arthritis. The results from the longitudinal phase of the study must be treated with caution because of the lack of a control group. Overall, the results of investigations of the ASMP in other countries are largely in accordance with those found in the US. However, improvements in pain have not been consistently demonstrated in RCTs.

Some questions remain about the generalisability of the findings with the ASMP especially to people with long-standing RA regularly using hospital services. The ASMP, was designed as a community-based programme for the mild to moderate end of the disease spectrum (Lorig & Holman, 1993). Results from studies of modified versions of the ASMP recruiting RA participants who are regularly using hospital services have been

less encouraging (Lindroth *et al.*, 1989; Taal *et al.*, 1993b). A further issue concerns the method of recruitment employed in most ASMP studies. Participants are invited to enrol in a self-management intervention. As such, they are likely to be a subset of the total population of people with arthritis, who are motivated to participate actively in the management of their arthritis. Motivational factors are critical to the success of psycho-educational interventions and it remains unclear how effective the ASMP would be for people with arthritis who are less motivated. Different recruitment strategies and research designs are needed in order to explore the influence of motivational factors in known populations with arthritis (Barlow *et al.*, 2000).

3.5.3 Cognitive behavioural interventions

With respect to CBT interventions there is now considerable evidence to suggest that they are effective treatments for RA. Studies have shown that CBT is effective in reducing depression, pain, disability and joint function (e.g. Applebaum, Blanchard, Hickling & Alfonso, 1988; Bradley *et al.*, 1987; O'Leary *et al.*, 1988; Young, 1992). Early studies of CBT for RA found conflicting results, and in a review, Young (1992) suggested that those studies with positive outcome all utilised broad based cognitive and behavioural interventions, which are aimed at symptom management. Those studies which used single component interventions (e.g. relaxation or thermal biofeedback) or that do not focus on the management of RA have generally shown poorer outcome (e.g. Mitchell *et al.*, 1986).

For example, Achterburg, McGraw & Lawlis (1981) and Mitchell *et al.*, (1986), both used thermal biofeedback as the primary therapeutic tool in their studies. Both studies found favourable results, with Achterberg *et al.*, (1981) reporting decreases in pain, muscle tension levels, sleep disturbance and increases in activities of daily living and Mitchell *et al.*, (1986) reporting improvements in pain and stiffness. However, in both studies a control group was included who were given reverse feedback. That is, the feedback indicated that their hand temperature had increased when it had really decreased and vice versa. Therefore, for the control groups they were actually learning to reduce peripheral circulation. Interestingly, the control groups also improved and there was no

difference between the two groups. This would suggest that it was non-specific factors that were likely to have brought about the changes that occurred rather than the ability to increase circulation.

Some other early studies found less than impressive results of cognitive and behavioural interventions applied to RA. Shearn and Fireman (1985) used a “stress management” intervention for people with RA. Again, while they found improvements in patient functioning, they also found improvements in the control group that were not significantly different. Strauss *et al.*, (1986) found even more disappointing results. They applied assertiveness and relaxation training with a group of RA patients and found that there were no improvements following treatments. Both these studies have been strongly criticised as they adopted a relatively conventional mental health format and failed to emphasize pain or symptom management as the primary goals of their interventions (Young, 1992). Indeed, Young (1992) notes that consistent with this criticism, both studies also reported high dropout rates. This may be an indication that the patients failed to see the relevance of the intervention and therefore were perhaps unlikely to benefit.

Studies of more comprehensive CBT interventions have shown more positive results. Bradley *et al.*, (1987) demonstrated the efficacy of a comprehensive cognitive behavioural approach to the management of chronic R.A. (mean duration 11.49 years). They found reductions in subjective pain, pain behaviours, anxiety and disease activity. Theirs was the first well-controlled study to demonstrate improvements over routine medical management and a social support (attention placebo) condition. The attention placebo intervention only resulted in reductions in trait anxiety at post-treatment and no improvements at six-month follow-up. Their treatment combined education with training in skills in thermal biofeedback, relaxation, behavioural goal setting and use of self-rewards. Their group also included family members. Young, Bradley and Turner (1995) report an 18-month follow-up of this cohort. They found that persons who had received the combined cognitive behavioural and biofeedback intervention had fewer RA- related clinic visits and days hospitalised, and lower overall costs of medical services, when compared with the social support control condition and no treatment control.

O'Leary *et al.*, (1988) used a cognitive-behavioural approach, incorporating features of the ASMP, for people with RA. The intervention had a major emphasis on increasing self-efficacy. Participants at post-treatment and four month follow-up demonstrated improvements in pain, joint function and self-efficacy. These improvements were greater than those gained by a control group who were given bibliotherapy. Interestingly, although O'Leary *et al.*, (1988) did not find significant changes in measures of disease activity following treatment, changes in ESR were associated with changes in self-efficacy.

Radojevic *et al.*, (1992) tested whether the addition of a family support component could enhance the efficacy of a behaviour therapy intervention for controlling RA symptoms. A sample of 65 persons with RA were randomly assigned to behaviour therapy alone, behaviour therapy with family support, arthritis education with family support, or a no treatment control condition. All treatments were carried out in small group sessions that met weekly for 6 weeks. Data analyses revealed that, when compared with the control conditions, the behavioural interventions showed significant improvements in number of swollen joints and severity of swelling post-treatment and at 2-month follow-up. These interventions also led to improvements in pain as assessed during direct joint examination at 2-month follow-up. The behavioural intervention with family support was superior to all other conditions combined on swelling measures at post treatment, but did not differ from the behaviour therapy without family support at follow-up. Radojevic *et al.*, (1992) concluded that cognitive– behavioural interventions are effective in managing RA disease-related symptoms and that involving family members may be helpful.

There is evidence that studies which have relied most heavily on people with severe, long-standing RA have found the least benefit from psychological interventions (Kraaimaat, Brons, Geenan & Bijlsma, 1995c; Parker *et al.*, 1988b). Studies of samples with shorter durations of RA seem to have found better results (Parker *et al.*, 1995; Sharpe *et al.*, 2001b).

For example, Parker *et al.*, (1988b) conducted a study comparing CBT to an attention placebo intervention with a group of predominantly male patients with moderate to severe disease. Although there were no changes in disease indices, improvements were noted in coping strategies and self-efficacy. Indeed, their results indicated that patients who were highly adherent to the protocol six months later, improved more with regard to all coping strategies assessed. Despite these improvements, neither six nor twelve month data supported improvements in pain, disease status or measures of psychopathology. Parker *et al.*, (1988b) explain their findings in the context of their sample characteristics. Their sample was predominantly male and was “limited in terms of education and income” (Parker *et al.*, 1988b) as well as having moderate to severe disease. Indeed, consistent with their explanation twelve-month follow-up indicated that all participants had deteriorated over time. It must be noted, however, that Parker *et al.*, (1988b) delivered the intervention in one week during an in-patient admission and follow-up assessments were taken six months later. It may be that an intensive in-patient treatment is less likely to generalise the results to a period when the patients return to their normal routine. Equally, it may be that results in the short-term were positive, but the benefits had not been maintained at six or twelve months.

These results were replicated by Kraaimaat *et al.*, (1995c) who conducted a study of 77 people with long-standing RA, in which they tested the relative efficacy of a cognitive-behavioural intervention and an occupational therapy intervention. Participants in the cognitive-behavioural intervention received training in progressive relaxation, rational thinking, active coping skills, and goal setting. Participants in the occupational therapy condition received educational information about biomedical aspects of RA, training in energy conservation, and exercises for reducing stiffness and increasing or maintaining joint mobility. Although persons in both conditions showed immediate improvements in knowledge about their disease, there were no improvements in health status. The cognitive-behavioural intervention produced post-treatment gains in pain coping. However, this was only on one of six coping strategies measured by the Pain Coping Inventory. The failure to find improvements was attributed to the finding that clinical and laboratory studies showed that this group of persons experienced significant deterioration

in their disease status over the course of treatment.

In contrast, Parker *et al.*, (1995) examined the efficacy of a cognitive behavioural stress management programme for people with RA within 4 years of diagnosis. Participants in the stress management programme received a very comprehensive protocol that featured a variety of components, including relaxation training, methods for identifying stressors and life goals, methods for managing pain, mood, and interpersonal relationships, and specific strategies for managing stressors typical of RA. An innovative feature of this protocol was that it integrated therapist training in coping skills with a computer- driven multimedia presentation. Another key feature was the use of booster treatment sessions scheduled at least once every 3 months over a year. At the end of treatment, results showed that, when compared with an attention control condition and a standard care control condition, persons in the stress management condition showed statistically significant immediate improvements in pain, coping, helplessness, self- efficacy, and health status. At the 15-month follow up, improvement in helplessness, coping and self- efficacy were still evident. This study showed that CBT can help people early in the course of RA to manage a variety of stressors associated with the illness.

However, it is important to point out, that this study varied in many other ways to their earlier work. For example, this was in a more traditional CBT format of delivery in the sense that it was conducted on an outpatient basis over a ten-week period. In addition, there was an additional 15-month “maintenance” phase. It remains unclear what the differential results are attributable to, the different mode of delivery or the different sample characteristics. Together these studies suggest that further research is needed to develop effective interventions for people with chronic RA.

3.5.4 Other psychosocial interventions

Although other psychosocial interventions have been investigated in only a few studies they do suggest some promising areas for further research. Telephone counselling has been associated with positive outcomes on measures of physical and psychological function (Maisiak, Austin & Heck, 1996). One study found improvement in depression in people with RA following non-directive group psychotherapy (Kaplan & Kozin, 1981). However, overall data on the effectiveness of psychotherapy and mutual support groups for individuals with rheumatic disorders is inconclusive (DeVellis & Blalock, 1993; Hawley, 1995). There is also evidence that interventions that involve family members and friends as an adjunct to other programs often have better outcomes (Lanza & Revenson, 1993). Finally, emotional disclosure of stressful events has been associated with improved psychological functioning (Kelley, Lumley & Leisen, 1997) and disease status parameters (Smyth, Stone, Hurewitz & Kaell, 1999) in people with RA.

3.5.5 Reviews and meta-analyses of psycho-educational interventions

The efficacy of psycho-educational interventions for rheumatic disease have been reviewed by a number of authors. There have been four meta-analyses (Astin *et al.*, 2002; Hawley, 1995; Mullen, Laville, Biddle & Lorig, 1987; Superio-Cabuslay *et al.*, 1996) and numerous narrative and systematic reviews (e.g. Boutaugh & Lorig, 1996; Bradley & Alberts, 1999; DeVellis & Blalock, 1993; Hirano *et al.*, 1994; Newman & Mulligan, 2000; Taal *et al.*, 1995).

There is consistent evidence that psycho-educational interventions can significantly increase knowledge about arthritis and practice of self-care behaviours such as exercise and relaxation (Hawley, 1995; Hirano *et al.*, 1994; Lorig *et al.*, 1987). For example, Lorig *et al.*, (1987) found that 32 of 34 (94%) studies showed increases in knowledge. They also reported that 37 of 48 (77%) studies showed increase practice of self-management behaviours (e.g. exercise, joint protection and relaxation). Hirano *et al.*, (1994) reviewed a further 25 psycho-educational intervention studies published after 1987. They found that 7

of 8 studies reported improvements in knowledge and for four the change was significant. The 25 studies included 34 measures of 12 self-management behaviours. There were positive changes in 29 of the measures and the changes were significant for 12 measures. No studies found negative changes in knowledge or self-management behaviours.

Reviews of psycho-educational interventions have shown that they can produce therapeutic benefits reducing pain, disability, depression, anxiety, tender joint count and work function (Boutaugh & Lorig, 1996; Hawley, 1995; Hirano *et al.*, 1994; Lorig *et al.*, 1987; Mullen *et al.*, 1987). However, not all studies showed improvements in health outcomes. Lorig *et al.*, (1987) reported that 46 of the 76 (61%) studies showed improved health outcomes.

Hirano *et al.*, (1994) reported that, in the 25 studies they reviewed, there were 52 measures of health status. Positive changes were found in 40 and changes were significant in 27. There were also 25 measures of nine psychosocial status variables. Positive changes were found in 20 and significant changes in 12. There were no negative changes in health status or psychosocial variables.

In the first published meta-analysis, Mullen *et al.*, (1987) reviewed 15 controlled trials of psycho-educational interventions for rheumatic disease. They found average additive reductions of 16% in pain, 22% in depression and 8% in disability. They concluded that psycho-educational interventions can contribute to improved patient outcomes in both RA and OA. Although as they noted, the pooled effect sizes were relatively small (0.20 for pain, 0.27 for depression, 0.09 for disability). The methodological quality of the trials was not assessed in this review.

Taal *et al.*, (1995) reviewed 31 studies of psycho-educational group interventions, 12 with OA or mixed diagnostic groups and 19 with only RA participants. Changes in knowledge were measured in 16 studies. Most studies that measured knowledge found significant short- and long-term beneficial effects of psycho-educational group intervention. Changes in self-management behaviours were measured in 16 studies. All studies that dealt with mixed populations, and measured self-management behaviour, demonstrated significant short- and

long-term beneficial effects. All of these interventions included some kind of behavioural training. Only a few of the group programs for people with RA found positive changes in self-management behaviour. However, several studies did not assess whether changes occurred in all behaviours that were taught.

Most studies, 27, measured one or more aspects of physical health status. Significant short-term improvements in health status were found in 66% of the studies that dealt with mixed populations and measured pain or other aspects of physical health status. Many also found long-term effects. Fewer of the psycho-educational interventions for people with RA demonstrated improvements in physical health status. Beneficial short-term effects were demonstrated in 47% of studies but only two studies found long-term effects.

Changes in psychosocial health status were assessed in 22 studies. In mixed populations, 3 of 7 studies found beneficial short-term changes and 3 of 4 studies found long-term benefits. Positive short-term changes in psychosocial health were found in 5 of 14 psycho-educational group intervention studies for people with RA. However, long-term benefits were only found in 1 of 8 studies.

Overall, this review shows that positive changes have been found more consistently in knowledge, self-management behaviours and physical health status. Positive changes in psychological health status are less common. They note that several studies have shown that people with rheumatic disease view the most problematic aspects of their condition to be pain, disability and dependence on others while far fewer report psychological problems such as depression or anxiety (Cornelissen, Rasker & Valkenburg, 1988; Lorig, Cox, Cuevas, Kraines & Britton, 1984; Van Lankveld, Naring, Van der Staak, Va't Pad Bosch & Van de Putte, 1993). Therefore, changes in psychological health status may be less relevant.

The review also suggests that psycho-educational group interventions for mixed rheumatic disease populations have more consistently demonstrated positive changes in self-management behaviours and long-term physical and psychological health status, than studies of RA populations. They suggest a number of possible reasons why psycho-

educational group interventions with RA samples may be less effective than those with mixed populations. First, the studies with mixed populations predominantly used comprehensive self-management interventions and the studies with RA samples used more focused CBT interventions that may have omitted key components. Second the average sample size in the studies of RA populations was smaller increasing the likelihood that clinically meaningful effects may not have been statistically evident, a Type II error. Third, as RA is a more serious condition, with higher levels of pain and disability, it is possible that it is more difficult to influence behaviour and health status in RA than in OA. The differences may also reflect the fact that the studies of mixed samples primarily used community samples and the CBT interventions used samples drawn from hospital clinics who are likely to have more serious disease. Studies testing more comprehensive self-management interventions with larger samples of people with RA drawn from hospital clinics may help to differentiate between these explanations.

Hawley (1995) carried out a meta-analysis of 34 psycho-educational intervention studies with 54 treatment arms. She notes that studies of psycho-educational interventions are difficult to compare because of the differences in design, participants, interventions, measures, follow-up times and variations in the quality of the studies. Overall, she found that 20 of 26 studies found improvements in pain and 15 of 20 studies found improvements in depression. The results for disability were less consistent. Most self-management interventions with OA or mixed diagnoses showed a small improvement but results of CBT or other interventions that were mainly conducted with RA samples showed no clear pattern. She suggests that it may be unrealistic to expect psycho-educational interventions to have a long-term effect on functional ability since it is expected that people with chronic rheumatic disorders will worsen over time. For example, follow-up studies of two groups of participants in self-management programmes at 20 months, reported declines in functional abilities of 4.8% and 2.5% (Lorig *et al.*, 1985; Lorig & Holman, 1989). At a 4-year follow-up of 401 participants of self-management groups, function had declined 9% (Lorig *et al.*, 1993). Another problem with interpreting functional change is that most functional questionnaires are correlated with psychological status. Thus, improvement in psychological factors or self-efficacy might lead to endorsement of better functional status

even when function does not change. No study has specifically tested functional ability by measuring performance of routine activities. Thus the effect of psycho-educational interventions on function remains unclear.

To put the benefits of psycho-educational interventions in context, Superio-Cabuslay *et al.*, (1996) compared a meta-analysis of 19 psycho-educational studies and a meta-analysis of non-steroidal anti-inflammatory drugs for people with rheumatic disease. As the primary focus of this meta-analysis was patient education, the authors chose to include only studies that had an information component and to exclude studies that were psychological or behavioural without a significant informational component. They found that psycho-educational interventions produced improvements in pain equivalent to 25%, in RA, and 20%, in OA, of the improvement produced by NSAIDs. In RA they also found 30-40% of the improvement in functional ability and 60-80% of the improvement in tender joint count found in the meta-analysis of NSAIDs. Compared with the NSAID studies, effect sizes for the studies of psycho-educational interventions for people with RA were fairly small (0.16 for pain; 0.18 for functional disability; 0.34 for tender joint count). However, the authors suggest that because most RA patients in these trials were already taking NSAIDs, the relatively small effect sizes probably represent the additional benefit over and above medication and may therefore be clinically relevant. The results suggest that psycho-educational interventions for RA can produce clinically significant improvements in pain, disability and tender joint count which are additional to the benefits of pharmacotherapy.

These meta-analyses of psycho-educational interventions have included studies of people with different rheumatic diseases. One interesting question is whether psycho-educational interventions are equally effective for people with different types of rheumatic disease. Two of the meta-analyses compared the effect sizes for studies of people with OA with those for people with RA and reached apparently contradictory conclusions (Hawley, 1995; Superio-Cabuslay *et al.*, 1996). Hawley (1995) compared 11 RA studies with four OA studies and found that the average effect size for OA studies was larger than that for RA studies (pain 0.44 vs. 0.13; Disability 0.28 vs. -0.16; depression 0.56 vs. 0.01). She

concluded that the evidence for the benefits of psycho-educational interventions for OA is stronger than that for RA. Superio-Cabuslay *et al.*, (1996) compared nine RA studies with ten OA studies. They found that the average effect sizes for pain relief were similar for OA and RA but there was more evidence for beneficial effects on disability in the RA studies (pain 0.16 vs. 0.18; disability 0.0 vs. 0.18). The most likely reason for these results is the difference in the studies reviewed. Although Hawley reviewed more RA trials, there was considerable overlap with the RA studies reviewed by Superio-Cabuslay *et al.*, (1996). However, Superio-Cabuslay *et al.*, (1996) included trials where the sample was predominantly OA but included other types of arthritis. The average effect sizes for pain relief in RA trials are similar in both meta-analyses. The better results for OA trials found by Hawley (1995) may reflect the selection of studies reviewed. Hawley (1995) reports average effect sizes for six community studies with samples of mixed diagnostic groups, mainly OA and RA, which had lower effect sizes than the OA studies (pain 0.21, disability 0.08 and depression 0.12). Differences between the meta-analyses may also reflect differences in their methodologies (e.g. the methods for calculating effect size and the time period over which improvements were assessed). Thus there is consistent evidence that psycho-educational interventions produce modest improvements in pain in RA but it is unclear whether in OA the benefits are of a similar magnitude or are greater. The results for disability are more difficult to explain. It is unclear whether psycho-educational interventions produce improvements in disability for either RA or OA. Further, it is unclear whether psycho-educational interventions are more effective in reducing disability in RA or OA. Finally, Hawley (1995) also reported that improvements in depression were greater for OA than for RA. The inclusion of educational interventions in both meta-analyses may have masked better results for trials of self-management and CBT interventions.

Another important question is whether certain types of psycho-educational intervention for people with RA are more effective than others. The Superio-Cabuslay *et al.*, (1996) meta-analysis showed that the effect sizes for educational interventions for OA and RA were smaller than for psycho-educational interventions (Pain 0.07 vs. 0.18; disability -0.19 vs. 0.05; Tender joint count -0.12 vs. 0.43). As mentioned above, this meta-analysis excluded

psychological interventions without an educational component and the psycho-educational studies were mainly of the ASMP and CBT. Both Taal *et al.*, (1995) and Hawley (1995)'s reviews suggest that improvements in health status were reported less consistently in studies of CBT with hospital outpatients with RA than in studies of self-management interventions in community mixed rheumatic disease populations. Conversely, as discussed above, Superio-Cabuslay *et al.*, (1996) found similar results for improvement in pain and larger effect sizes for disability in studies of RA populations, predominantly educational and CBT interventions, than in studies of OA and mixed populations, predominantly educational and self-management interventions. However, it is not possible to determine whether these results reflect differences in the interventions, samples or other factors.

A recent meta-analysis has specifically reviewed randomised controlled trials of psychological interventions for people with RA (Astin *et al.*, 2002). This meta-analysis is particularly helpful because it used strict methodological quality criteria to select studies for review. They only included studies with an active psychological treatment component and excluded intervention studies that simply provided information. Only studies with purely RA samples or studies that included mixed diagnostic samples that reported separate data for RA were examined. This review is interesting as it included studies of more comprehensive CBT programmes and some novel psychological interventions that have been developed more recently. They reviewed 25 studies that met the methodological criteria. Of these, 13 studies (52%) (Appelbaum *et al.*, 1988; Bradley *et al.*, 1987; Germond, Schomer, Meyers & Weight, 1993; Kraaimaat *et al.*, 1995c; Liebing, Pfingsten, Bartmann, Rueger & Schuessler, 1999; O'Leary *et al.*, 1988; Parker *et al.*, 1988b; Parker *et al.*, 1995; Radojevic *et al.*, 1992; Scholten *et al.*, 1999; Sharpe *et al.*, 2001b; Shearn & Fireman, 1985; Taal *et al.*, 1993b) were either described by the researchers or could be characterized as multimodal, cognitive-behavioural interventions. These interventions typically involved some combination of relaxation, imagery, stress management, or the teaching of cognitive coping skills. Five studies (Appelbaum *et al.*, 1988; Bradley *et al.*, 1987; Flor, Haag, Turk & Koehler, 1983; Lavigne, Ross, Berry, Hayford & Pachman, 1992; Van Deusen & Harlowe, 1987) also included biofeedback as

one of the treatment components. Five studies employed more traditional psychotherapeutic interventions, both group based (Kaplan & Kozin, 1981; Lindroth *et al.*, 1997) and individual (DeVellis, Blalock, Hahn, DeVellis & Hochbaum, 1988; Maisiak *et al.*, 1996; Maisiak, Austin, West & Heck, 1996), and the intervention in two studies (Kelley *et al.*, 1997; Smyth *et al.*, 1999) involved a novel emotional disclosure protocol in which participants were asked to write or speak about difficult emotional or stressful experiences. Length of the interventions varied from 3 days to 9 months with a mean of 9.8 weeks. One study (Parker *et al.*, 1995) utilized a refresher course following the actual intervention. Nineteen of the 25 studies collected follow-up data with follow-up time periods ranging from 2 to 18 months (mean of 8.6). Effect sizes were calculated for six outcomes (see Table 3.1). Significant pooled effect sizes were found post intervention for pain (0.22), functional disability (0.27), psychological status (0.15), coping (0.46), and self-efficacy (0.35). At follow-up (averaging 8.5 months), significant pooled effect sizes were observed for tender joints (0.33), psychological status (0.30), and coping (0.52).

Table 3.1 Pooled effect sizes for six outcomes from a meta-analysis of psychological interventions for RA (Adapted from Astin *et al.*, 2002)

	Effect Sizes	
Study Outcomes	Post Intervention	Follow-up
Pain	0.22	0.06
Disability	0.27	0.12
Tender Joints	0.14	0.33
Psychological Status	0.15	0.30
Coping	0.46	0.52
Self-Efficacy	0.35	0.20

Although statistically significant pooled effect sizes were found for most of the outcomes, these effects were relatively small. In addition, at the individual study level the results

were less consistent. The majority of trials failed to show significant treatment effects on the 6 outcomes and a number of studies actually showed a negative treatment effect. Further, the long-term effects of psychological interventions are unclear, while some benefits appear to diminish over time (e.g. pain, disability others appear to be more pronounced (e.g. psychological status, tender joint count). No clear or consistent patterns emerged when effect sizes for different types of treatment and control conditions were compared, or when higher quality trials were compared to lower quality ones. One key finding was that these psychological interventions may be more effective for people who have had RA for a shorter duration. Studies with samples with longer illness duration had lower effect sizes than studies with shorter illness duration (pain, 0.19 vs. 0.46; disability, 0.33 vs. 0.45; psychological status, 0.08 vs. 0.34; and coping, 0.43 vs. 0.49. These analyses should be interpreted with some caution, however, because of the smaller number of trials constituting each of the pooled effect sizes.

Although all the studies reviewed met stringent criteria for methodological quality, they did note some methodological weaknesses in some studies which may have biased the results including: small sample size and possibly lack of statistical power; little use of allocation concealment methods; failure to use intent-to-treat analyses; lack of control for multiple comparisons and under reporting of non-significant findings. They conclude that despite methodological flaws in the literature, psychological interventions may be important adjunctive therapies in the medical management of RA.

An important question that has not been fully answered by the research is the extent to which the effects of psycho-educational interventions produce long-term effects. Keefe and Van Horn (1993) reviewed the long-term effects of psycho-educational interventions for RA. They conclude that although psycho-educational interventions often result in reductions of pain and disability in the short-term, the majority of studies have failed to demonstrate maintenance of treatment gains at follow-up. Many studies have failed to measure long-term effects. For example, Taal *et al.*, (1995) found that 16 of 31 studies measured no long-term effects after four months.

Taal *et al.*, (1995) report that although few studies of RA populations had found long-term effects on physical health status positive results were often found in studies of mixed populations. For example, for the ASMP Lorig *et al.*, (1985) report that improvements in knowledge, behaviours and pain remained significant at 20 months. Lorig and Holman (1989) report that at 20-month follow-up there was a 20% reduction in pain, 14% reduction in depression and 35% reduction in doctor visits compared to baseline. At a 4-year follow-up, Lorig *et al.*, (1993) found that pain scores were 20% lower than baseline but disability had increased by 9%. Despite this depression had not increased and there was a 40% decrease in doctor visits.

However, these studies used a waiting list control group design, in which participants in the control group received the intervention after short-term follow-up. In these studies long-term results are based on within group analyses. This is a significant problem for interpretation of the longitudinal phase of ASMP studies, especially given the recruitment methods usually employed in these studies. People may be more likely to volunteer to attend a self-management programme when they are experiencing more difficulty managing their arthritis. Enrolment during periods of heightened disease activity or severity might influence the apparent effect size of the intervention. In non-controlled studies regression to the mean may produce apparent treatment effects when, in reality, the effect of treatment is small or non-existent. How strong such a factor might be is uncertain, but as much as 30% of the effect, based on data from randomised controlled trials and longitudinal observational studies of drug treatment interventions, could be explained by regression to the mean (Hawley, 1995).

One study has investigated the efficacy of self-management intervention for people with RA using a methodology that allowed comparison between treatment and control group at long-term follow-up. Taal *et al.*, (1993b) evaluated a modified version of the ASMP delivered by health professionals for groups of hospital outpatients with RA, mean age 60 years and duration 14 years. They found at 14-month follow-up that improvements in disability seen at 4 months were not maintained. However, effects on knowledge, self-efficacy and the practice of physical exercises were still evident.

A few studies of CBT for people with RA have shown long-term changes in health outcomes (e.g. Bradley *et al.*, 1987). However, consistent with Taal *et al.*, (1995), Astin *et al.*, (2002) found in their review of psychological interventions for RA that for some outcomes (pain and disability), treatment effects appeared to diminish over time, becoming statistically non significant at follow-up. However, in the case of psychological status (depression) and tender joints, effects became somewhat more pronounced over time. These findings suggest that more work is needed to identify effective relapse prevention strategies. Future trials should explore the potential value of building booster/relapse prevention strategies into the trial designs, as was done by Parker *et al.*, (1995) in their study.

In summary, in contrast to educational interventions, more comprehensive and action-oriented interventions (like CBT) and self-management programmes have been associated with a variety of positive outcomes for people with rheumatic disease (Boutaugh & Lorig, 1996; Bradley & Alberts, 1999). In RA, short-term improvements have been found most consistently for pain but less consistently for disability and depression. The long-term effects of psycho-educational interventions on health outcomes are unclear. Despite this, CBTs and the ASMP have satisfied the American Psychological Association's criteria for empirically validated treatment for pain among RA and OA populations (Compass, Haaga, Keefe, Leitenberg & Williams, 1998). The criteria for well-established interventions, as set forth by the American Psychological Association, include a demonstration of efficacy through at least two controlled outcome studies from different investigators. Given the positive findings for pain reduction, further investigations of psycho-educational interventions for RA are warranted (McCracken, 1991; Parker *et al.*, 1993). Additional research is needed to clarify which psychological interventions (or combinations of interventions) are most effective and for which specific types of patients and outcomes.

3.6 Mechanisms of change in psycho-educational interventions

The average effect sizes for psycho-educational interventions found in meta-analyses are modest and many studies have failed to demonstrate long-term benefits (Astin *et al.*, 2002; Hawley, 1995; Mullen *et al.*, 1987; Superio-Cabuslay *et al.*, 1996; Taal *et al.*, 1995). There are also large variations in the results of studies and, as with the results of studies of educational interventions, the results of different studies of self-management and CBT interventions have been somewhat conflicting. A better understanding of the mechanisms of change may lead to the development of interventions with improved immediate and long-term outcomes.

There is converging evidence supporting the view that cognitive and behavioural factors may be important proximal variables mediating between psycho-educational intervention and health outcome benefits. The research reviewed, in Chapter 2, suggests that cognitive representations of illness, coping behaviours, and appraisals are associated with pain, disability, and emotional well being concurrently and prospectively. Several studies have also examined the effect of psycho-educational interventions on cognitive and behavioural factors but relatively few studies have directly manipulated cognitions or coping behaviours to investigate whether they mediate the therapeutic benefits of psycho-educational interventions.

The literature suggests that appropriate re-structuring of the person's illness representation is necessary if self-regulation of pain, disability, and emotional distress is to be improved. Interventions may fail to show benefits because they produce unhelpful changes in illness representations. In one study Parker *et al.*, (1984) presented RA patients with a seven-hour educational programme. Although patients' knowledge about the disease increased, the intervention group reported more pain and disability than the control group. The presentations may have had a negative influence on illness cognitions by highlighting the salience of pain and the possible negative effects of RA. People make their own interpretations of their experiences in psycho-educational interventions and these are not always those anticipated by health professionals. It is important to consider how an

intervention will affect people's illness representations and how such a change will lead to improvements in the person's ability to successfully self regulate the illness.

There is some evidence that psycho-educational interventions produce adaptive changes in cognitions and coping behaviours. Goeppinger, Arthur, Baglioni, Brunk & Brunner (1989) compared a self-management intervention delivered either as a group programme or a home study package with a control group in 374 people with arthritis. The intervention groups had significant improvements in arthritis knowledge, self care behaviours, pain and modest decreases in learned helplessness scores, which persisted for 12 months. Changes in learned helplessness and self care behaviours appeared to account in part for improvements in pain. Parker *et al.*, (1995) also reported significant reductions in helplessness following a stress-management intervention for RA, which persisted at 15-month follow-up. Parker *et al.*, (1988b), primarily studying elderly men with RA, report that participants in a cognitive behavioural pain management programme catastrophized significantly less and reported increased perception of control over pain compared to attention placebo or no treatment control groups. Although the cognitive behavioural intervention produced changes in catastrophizing and beliefs about pain controllability, it did not produce therapeutic benefits in pain or depression. In a subsequent longitudinal study Parker *et al.*, (1989) report that changes in catastrophizing and beliefs about pain controllability were associated with changes in pain, physical function, and arthritis helplessness. These studies suggest that changes in catastrophizing and beliefs about pain control are important but not sufficient to produce improvements in health status.

Several studies of people with RA have demonstrated significant changes in coping following CBT (Kraaimaat *et al.*, 1995c; Liebing *et al.*, 1999; O'Leary *et al.*, 1988; Parker *et al.*, 1988b, 1995; Radojevic *et al.*, 1992; Scholten *et al.*, 1999).

Radojevic *et al.*, (1992) found that behaviour therapy, behaviour therapy with family support, and education with family support interventions for RA all produced increases in the use of active coping strategies compared to a no treatment control group. However these differences were not evident at 2-month follow up. They also found decreases in the use of

passive coping strategies in all intervention groups at two-month follow-up. The changes in active and passive strategies were greatest in the behaviour therapy without family support group.

Another, more recent, study of people with RA has shown changes in coping. Liebing *et al.*, (1999) compared the effects of a comprehensive cognitive– behavioural treatment protocol with a routine care control condition. When compared with people in the control condition, people in the cognitive– behavioural treatment condition showed significant improvements in pain affect, coping, and emotional stability. Scholten *et al.*, (1999) conducted a 1-year RCT and a 5-year longitudinal study of a health professional led psycho-educational group intervention for people with RA. They found improvements in coping, assessed on the Freiburg Questionnaire of Coping with Illness, at the 1-year follow-up of the RCT and at the 5-year follow-up of the longitudinal study.

Lorig and her colleagues have conducted a series of studies to establish which variables are associated with the benefits of the ASMP (Lorig & Gonzalez, 1992). The benefits were not mediated by changes in performance of self-management behaviour as they were only weakly associated with changes in health status (Lorig *et al.*, 1989b). Rather, changes in self-efficacy (for pain and other symptoms) were found to be particularly important factors in the outcome of self-management intervention. Changes in self-efficacy (for pain and other symptoms) following the ASMP were strongly associated with improvements in health status (pain and depression) (in the short-term Lorig *et al.*, 1989b) and even up to 4 years later (Lorig *et al.*, 1993).

Several other studies have found positive changes in self-efficacy following psycho-educational interventions (Davis, Busch, Lowe, Taniguchi & Dikowich, 1994; Liebing *et al.*, 1999; Lorig & Holman, 1989; O'Leary *et al.*, 1988; Parker *et al.*, 1995; Taal *et al.*, 1993b). Smarr *et al.*, 1997 report predictors of outcome from the Parker *et al.*, 1995 stress-management intervention study for people with RA. Smarr *et al.*, (1997) report that increases in self-efficacy over treatment were related to improvements in pain, depression, health status and disease activity.

However, as Lorig and Gonzalez (1992) note, these studies do not prove that enhancing self-efficacy improves health status. It is possible that a third factor may produce change in both self-efficacy and health status, self-efficacy may influence health status through a third factor, or improved health status may enhance self-efficacy. As Sharpe, Sensky and Allard (1999) stated it is unclear whether improved health status is related to objective increases in control or improved perceptions of control or a combination of both.

To test these hypotheses Lorig and her colleagues redesigned the ASMP to enhance participants' self-efficacy beliefs, introducing strategies such as modelling and behavioural contracts. They reported that this revised course produced greater changes in self-efficacy and health status, (Lorig & Gonzalez, 1992).

Thus changing self-efficacy or confidence in one's ability to manage the illness, seems to be an important mechanism by which psycho-educational interventions assist people to improve self-regulation of rheumatic disease. New guidelines for the management of pain in arthritis emphasize the importance of helping people build confidence in their self-help abilities as a routine part of the biomedical and psychosocial treatment process (Simon *et al.*, 2002).

A recent study has examined how illness representations might influence the efficacy of CBT for people with early RA. In this study 53 patients having RA for less than 2 years were randomly assigned to a CBT intervention or routine medical treatment control condition (Sharpe *et al.*, 2001b). Analyses revealed that at 6-month follow-up, patients in the CBT group had significant decreases in depression and in the number of actively inflamed and painful joints, and increases in the use of the coping strategies of attention diversion and reinterpreting pain. Sharpe *et al.*, (In press) reported on the 23 participants who received the CBT intervention. Illness representations were assessed using the IPQ. Pre-treatment indices and demographic variables were investigated as predictors of response in terms of mood at post-treatment. Changes in variables over treatment were investigated as predictors of changes in joint function between the end of treatment and

follow-up. People with initially high levels of disability and who believed the illness not to be very serious showed less improvement in depression at follow up. Changes in anxiety and depression over the treatment period predicted subsequent changes in the level of joint functioning between post treatment and follow-up. These results suggest that acceptance of illness may be an important process in maintaining positive mood following diagnosis. Positive mood, in turn, predicted improvements in joint function six months later, confirming that changes in psychosocial factors can impact on physical parameters of disease.

In summary there is considerable evidence that key components of the self-regulatory model play a crucial role in the outcome of psycho-educational interventions. However the relative importance of cognitive representations, coping efforts, and appraisals in the outcome of psycho-educational intervention is unclear. Further the precise mechanisms by which psycho-educational interventions enable people to successfully manage the consequences of rheumatic disease are poorly understood.

Studies based on psychological theory have led to the development of the most successful psycho-educational interventions (DeVellis & Blalock, 1993, Hirano *et al.*, 1994). However no study has explicitly used the self-regulatory model to guide investigation of how illness cognitions, coping behaviours, and appraisals may interact during psycho-educational intervention for people with chronic RA.

Psycho-educational intervention can be viewed as a method for experimentally manipulating self-regulatory processes. Studying the interaction between self-regulatory variables over the course of a psycho-educational intervention may provide insight into the dynamic processes by which a person can enhance their self-management of chronic rheumatic disease. This in turn could lead to the development of improved psycho-educational interventions.

Intervention studies also provide an opportunity to test the self-regulatory model. Such studies enable us to investigate whether the self-regulatory model provides a useful framework for understanding self-management of rheumatic disease. Studies are needed to

investigate whether the self-regulatory model provides useful insights into the processes by which self-management interventions assist people with RA to improve their health outcomes.

3.7 Summary and future research

Psycho-educational interventions have been shown to improve knowledge, self-management behaviours adaptive cognitions and less consistently to reduce pain, disability depression and health-care utilisation. However, the average effect sizes for psycho-educational interventions found in meta-analyses are modest and many studies have failed to demonstrate long-term benefits. It is not fully clear who is likely to benefit from what type of intervention and at what point in their illness. The evidence reviewed suggests that self-management interventions are effective for people with arthritis recruited from the community and who wish to enrol on a self-management programme. CBT has been shown to be effective for people with OA and people with RA, especially early RA, recruited from hospital populations. However, it is unclear whether diagnosis, type of intervention or other factors explain the differences identified.

Further research is needed to investigate what factors (e.g. gender; diagnosis; disease severity; motivation to engage in self-management) influence response to treatment. An interesting question is at what point in the illness process should psycho-educational interventions be offered. There is little information regarding the time in the disease course when self-management may be optimally effective (Barlow *et al.*, 2002b). However, for psychological interventions for people with RA there is evidence that they may be less effective for those who have longer disease duration or more severe disease (Astin *et al.*, 2002). It has been recommended that psycho-educational interventions should be offered to people early in the course of RA (Sharpe *et al.*, 2001b). However, as many people are living with chronic RA and maintenance of treatment gains is problematic, effective interventions for people with chronic RA are needed. Given the relative lack of effectiveness of CBT interventions with chronic RA further research is

needed to develop interventions for this group. The potential of the ASMP for hospital outpatients with chronic RA needs further exploration.

The review suggests that the most successful interventions are comprehensive multiple component programmes. In particular, those that have been developed based on an understanding of the needs and beliefs of participants and that emphasise the reduction of pain disability and emotional distress. Using psychological methods in interactive small group settings to develop and maintain adaptive cognitions (e.g. self-efficacy), coping strategies (e.g. problem solving) and self-management behaviours (e.g. exercise) seem to be helpful. However, it remains unclear which components of these treatment packages are necessary. Interventions might also benefit from being tailored to suit participants (e.g. for a specific diagnosis, need, culture or point in the lifespan). A further area concerns the comparative effectiveness of different methods of delivering psycho-educational interventions especially those that have cost implications. Should interventions be: led by health professionals or people with arthritis; for individuals or groups; for a single or mixed diagnostic group; include family members; held in hospital or community settings; make use of new technology (e.g. the internet).

There is emerging evidence that the outcomes of psycho-educational interventions for rheumatic disease are related to changes in self-regulatory processes (e.g. cognitions and coping strategies). Further work is needed to explore the nature of these mechanisms to improve the efficacy of these interventions, in particular for people with chronic RA. The SRM may provide a useful framework for guiding this research.

Chapter 4: Summary of the literature and implications for the present study

4.1 Clinical context for the study

RA is the most common form of inflammatory arthritis, affecting millions of people worldwide. It is chronic, progressive, incurable and the prognosis is uncertain. It follows an unpredictable course of exacerbation and remission of inflammation with gradual progression of joint damage. RA is associated with excess mortality, distressing symptoms, moderate to severe functional disability, increased risk of depression and major social and economic consequences. The costs of RA to the person, health services and society are enormous. Current bio-medical approaches to treatment are ameliorative, aiming to reduce inflammation, relieve pain and maintain or improve function. However, they lack prolonged efficacy, are frequently associated with serious side-effects and fail to address the psychosocial needs of people with RA.

There is growing awareness of the need to incorporate psychosocial approaches into ongoing biomedical treatment for managing RA (Simon & Yocam, 2000). Increasingly treatment for RA is multifaceted and directed not only at immunological abnormalities, physical symptoms and disability but also at the social and psychological functioning of the patient (Keefe *et al.*, 2002).

4.2 The efficacy of self-management intervention for people with chronic RA

Self-management and cognitive-behavioural interventions for people with rheumatic disease have been shown to improve knowledge and self-management behaviours and, less consistently, to reduce pain, disability, depression and health-care utilisation (Huyser &

Parker, 2002). However, it is not fully clear who is likely to benefit, from what type of intervention and at what point in their illness. The evidence reviewed suggests that self-management interventions are effective for people with arthritis recruited from the community and who wish to enrol on a self-management programme (Barlow *et al.*, 2002b). CBT has been shown to be effective for people with OA and RA, especially early RA, recruited from hospital populations (Astin *et al.*, 2002; Hawley, 1995).

There is little information regarding the time in the disease course when self-management may be optimally effective (Barlow *et al.*, 2002b). However, for psychological interventions for people with RA, there is evidence that they may be less effective for those who have longer disease duration or more severe disease (Astin *et al.*, 2002). As many people are living with chronic RA, and maintenance of treatment gains is problematic, further research to identify effective interventions for people with chronic RA is needed. Given the relative lack of effectiveness of CBT interventions with chronic RA, the efficacy of the ASMP for hospital outpatients with chronic RA needs further exploration.

This study addresses this need for further research by investigating whether self-management intervention for hospital outpatients with chronic RA produces significant changes in health outcomes (Hypothesis 1.1). The study evaluates whether findings from previous studies of self-management intervention can be generalised to a UK sample of people with chronic RA routinely using hospital services. Previous studies of self-management interventions have mainly used waiting list control designs with follow-up at 4 months. The design of this study extends this research by testing whether differences between the treatment and control group are maintained at 9-month follow-up.

Previous research has shown that psycho-educational interventions can produce adaptive changes in cognitions (e.g. helplessness and self-efficacy) and coping behaviours (Astin *et al.*, 2002). This study aims to replicate and extend this research by examining whether a self-management intervention changes illness representations and coping procedures (Hypothesis 1.2).

4.3 The relationship between illness representations coping and health outcomes in chronic RA

There are considerable individual differences in the levels of pain, disability and emotional distress experienced by people with RA. Psychosocial factors usually account for a greater proportion of the variance in self-reported pain, disability and depression than objective measures of disease activity (Young, 1992). With no cure for RA, and with medical treatments only being partially effective, it is important to explore the self-regulatory processes that mediate between the disease, pain, disability, and psychological adjustment. Understanding how some people successfully make sense of and manage RA, minimizing pain and disability and optimizing psychological adjustment, may help in the development of interventions to assist people who are managing relatively poorly.

Leventhal's SRM has been found to be a useful framework for understanding adaptation to acute and chronic illness (Petrie & Weinman, 1997), however, only a few studies in RA have been guided by this model. In early and chronic RA, cognitive representations of illness and coping procedures have been associated with health outcomes in both cross-sectional (Murphy *et al.*, 1999; Scharloo *et al.*, 1998) and longitudinal studies (Scharloo *et al.*, 1999; Sharpe, 1999). The most consistent findings are that poorer physical and psychological adaptation and greater use of health services are associated with:

1. Perceiving RA as having a stronger illness identity, more serious consequences and to be more difficult to control;
2. Greater use of passive and less use of active coping strategies.

Although these findings are interesting, there have only been three studies and these have significant methodological weaknesses (e.g. small sample size, cross-sectional design or only examining a limited number of illness representations and coping procedures).

This study replicates previous research in a larger sample. In the cross-sectional phase, it tests whether illness representations and coping procedures are associated with health outcomes at baseline (Hypothesis 2.2) and, in the longitudinal phase, whether initial illness representations and coping procedures predict future health outcomes at 3, 6 and 12 months

(Hypothesis 2.3). This also allows an assessment of whether previous research can be generalised to a UK sample of people with chronic RA.

The complex dynamic relationships between self-regulatory processes and health outcomes are difficult to disentangle. Longitudinal designs assessing self-regulatory processes and health outcomes at multiple time points may elucidate these relationships. Previous research has not investigated whether changes in illness representations and coping influence future health outcomes in chronic RA. This study extends previous work by examining whether changes in illness representations and coping over time predict future health outcomes (Hypothesis 2.4). This allows a closer examination of the dynamic processes of self-regulation proposed by the SRM.

4.4 The role of illness representations and coping in moderating or mediating changes in self-management intervention

There is emerging evidence that the outcomes of psycho-educational interventions for rheumatic disease are related to changes in self-regulatory processes (Pimm & Weinman, 1998; Sharpe, 1999). Further work is needed to explore the nature of these mechanisms to improve the efficacy of these interventions, especially for people with chronic RA. The SRM may provide a useful framework for guiding this research.

In addition, an experimental manipulation of illness representations and coping procedures, through a psycho-educational intervention, may provide an opportunity to explore the complex dynamic processes involved in self-regulation of RA (Scharloo *et al.*, 1999). This study explores these questions by examining whether illness representations and coping procedures moderate (Hypothesis 3.1) or mediate (Hypothesis 3.2) the outcomes of self-management intervention for people with chronic RA.

4.5. Research questions

The research questions were formulated to provide a description of the outcome measures and self-regulatory variables in this sample and the way in which they change over time. This provides a context for the main aims and hypotheses.

RQ1.1 What are the baseline levels of disease activity, pain, physical function and emotional distress in this sample of people with RA?

RQ1.2 What are the illness representations of a sample of people with RA?

RQ1.3 Which coping strategies do the sample of people with RA use?

RQ1.4 How do health outcomes in a sample of people with RA change over one year?

RQ1.5 How do illness representations and coping procedures in a sample of people with RA change over one year?

4.6 Aims and hypotheses

4.6.1 The efficacy of self-management intervention for people with chronic RA

Aim 1: To investigate whether a RA self-management intervention produces therapeutic benefits in a UK sample.

Hypothesis 1.1 The intervention will produce a significant improvement in the following health outcomes immediately after intervention, at 3 and 9-month follow-up:

- a. Pain
- b. Physical disability
- c. Emotional distress
- d. Social/Occupational Function
- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

Hypothesis 1.2 The intervention will not produce any significant change in the following illness representations and coping procedures:

- a. Illness identity
- b. Symptom coherence
- c. Time line
- d. Consequences
- e. Cause
- f. Control/cure
- g. Self-efficacy
- h. Coping strategies
- i. Self-management behaviours

4.6.2 The relationship between illness representations coping and health outcomes in chronic RA

Aim 2: To use Leventhal's self-regulatory model to explore the processes by which people perceive and respond to RA.

Hypothesis 2.1 At baseline the following illness representations will be positively associated with greater use of adaptive coping (*acceptance, active coping, positive reinterpretation/growth and planning*) and less use of maladaptive coping (*behavioural and mental disengagement, denial and focusing on and venting emotions*):

- a. Lower Illness Identity
- b. Greater symptom coherence
- c. Less serious perceived consequences
- d. More chronic timeline
- e. Greater perceived controllability
- f. Less perceived causes

Hypothesis 2.2 Illness representations and coping procedures will account for a significant amount of variance in the following health outcomes in the pooled sample (independent of that accounted for by experimental condition) at time 1:

- a. Pain
- b. Physical disability
- c. Emotional distress
- d. Social/Occupational Function
- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

Hypothesis 2.3 Baseline illness representations and coping procedures will predict a significant amount of variance in the following health outcomes in the pooled sample (independent of that accounted for by experimental condition) at time points 3-5:

- a. Pain
- b. Physical disability
- c. Emotional distress

- d. Social/Occupational Function
- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

Hypothesis 2.4 Change in illness representations and coping procedures will explain a significant amount of variance in health outcomes in the pooled sample at time points 3-5:

- a. Pain
- b. Physical disability
- c. Emotional distress
- d. Social/Occupational Function
- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

4.6.3 The role of illness representations and coping in moderating and mediating outcomes of a self-management intervention

Aim 3: To investigate whether the outcomes of a RA Self-Management intervention are moderated or mediated by illness representations and coping procedures.

Hypothesis 3.1 Baseline illness representations and coping procedures will moderate the effect of the self-management intervention on health outcomes post treatment and at 3 and 9-month follow-up.

Hypothesis 3.2 Changes in illness representations and coping procedures will mediate the effect of the self-management intervention on health outcomes post intervention and at 3 and 9-month follow-up.

Chapter 5: Method

5.1 Design

The study design is a modified single blind randomized controlled clinical trial based on Zelen (1979). Figure 5.1 shows the design for the standard RCT and the modified Zelen's RCT. This is a two group (treatment and control) prospective design with dependent and independent variables measured at five time points. The study compares the effects of a self-management intervention plus standard medical care with standard medical care.

The design allows for the testing of hypothesis 1.1-2 through comparisons for each dependent (outcome) variable within subjects (time) at baseline, pre, post and 3 and 9-month follow-up, and between subjects (treatment or control). Repeated measures ANOVA was used to make the comparisons. Analysis of covariance was used if the two groups differed on critical variables at baseline. Hypothesis 2.2-4 were tested using multiple regression analyses to identify those variables (e.g. demographic, disease, psychosocial) which explain the outcome variables (e.g. pain, disability and emotional distress) in the pooled sample. Hypothesis 3.1-2 were tested using multiple regression analysis to identify whether illness representations and coping variables moderate or mediate the effects of the intervention on outcome.

5.2 Procedure

A total of 136 outpatients with RA were randomly allocated to treatment or control groups. The treatment group was offered a self-management group. All participants were assessed at baseline (0 weeks), pre-intervention (6 weeks), post intervention (12 weeks), 3-month follow up, (6 months), and 9-month follow-up, (one year). Assessments included a battery of self-report questionnaires, tender joint score and blood tests. See below for further details of procedure.

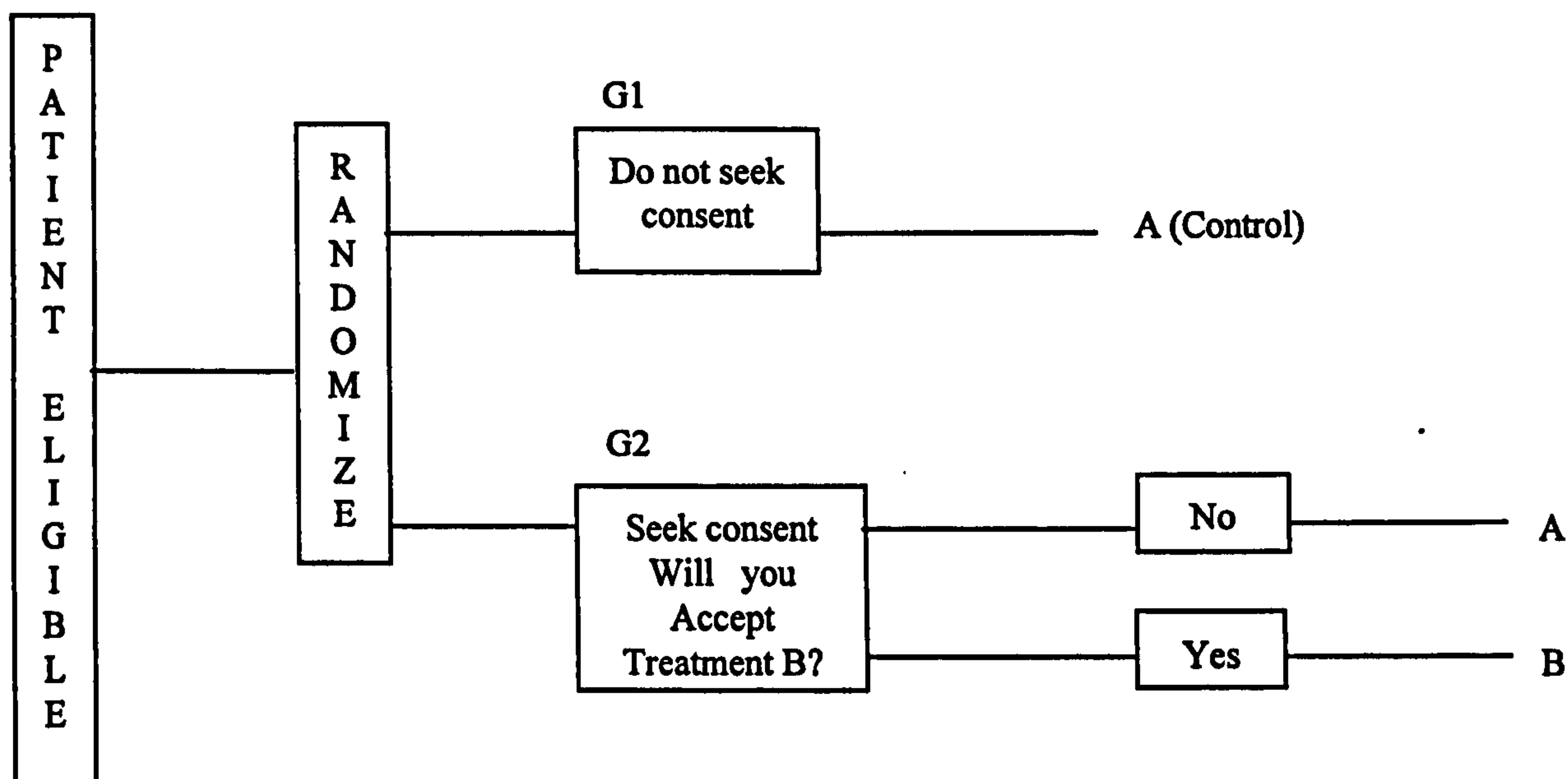


Figure 5.1: Zelen's modified design for randomised clinical trials. Patient is asked if new treatment is acceptable after both are discussed. (Zelen, 1979).

5.3 Participants

Participants were recruited in batches, before each self-management group, from consecutive attendees at Rheumatology outpatient clinics at Stoke Mandeville Hospital.

There were explicit inclusion and exclusion criteria for the study (Appendix D). At study entry a Rheumatologist had to confirm that each participant had a diagnosis of rheumatoid arthritis, as defined by the American Rheumatism Association 1987 Revised criteria (Arnett *et al.*, 1988; Appendix A). All participants had to have established disease of at least one year and be aged 16-70 years. They had to be able to attend five hospital appointments and six weekly group sessions over one year. In addition, they had to have reasonable understanding of English and no history of severe mental illness. Finally, they must not have participated in the pilot groups.

All participants were invited to take part in a study to investigate how people cope with RA. Participants were then randomly allocated to treatment or control groups. After randomisation, participants in the treatment group were then offered the opportunity to participate in the self-management group. A table of two-digit random numbers was used to generate a list of 150 group assignments before initial recruitment. As the study design meant that participants in the treatment arm of the study could decline to participate in the intervention the randomisation procedure allocated a larger number of participants to the treatment arm (45% control 55% treatment). Randomised blocks assignment of matched pairs which ensures relatively comparable groups was not used because of the difficulty in matching pairs of participants using batch recruitment and the size of the groups makes it unlikely that a serious imbalance would occur. Basic information was recorded about all participants who did not wish to participate in the study.

Sample size calculations were computed for two of the primary end points: pain and functional ability (Kraemer & Thiemann 1987). Calculations were based on the most recent data, available at the start of the study, from the Arthritis Self-Management

Programme (Holman & Lorig, 1992). For a one tailed test at the 5% significance level with an 80% power they showed the following numbers of participants would be sufficient to detect a significant difference: pain $N = 45$ and disability $N = 152$.

Holman and Lorig's (1992) data probably underestimate the potential for change in disability in the current study of RA patients, as a large proportion of their sample had osteo arthritis and were therefore less likely to show significant changes in functional ability. This is supported by Tucker and Kirwan's (1991) evaluation of an in-patient education group for people with RA which found improvement in functional ability despite the small sample size. Data on 25 participants who attended the pilot self-management course showed improvement in functional ability (Pimm, Amos, & Byron, 1992). Therefore it was estimated that a sample size of 120 participants [$n=60$ in each group] would be sufficient to detect improvements in pain and functional ability.

It was not anticipated that there would be any difficulty in obtaining sufficient numbers of participants for the study. From January 1991 to March 1992 there were 2074 referrals to the Rheumatology service. Approximately 15% of these had RA, suggesting an estimated 240 new RA referrals per year. During the same period 8692 people attended follow-up clinics. Approximately 60% had RA and they attended on average every 4 months, suggesting an estimated 1300 people with RA, per year, attended the follow-up clinics.

5.4 Assessment process

Participants were assessed at the point of recruitment (baseline assessment=0 weeks); after a 6 week baseline or "wash-out" phase (pre-intervention assessment=6 weeks); after the 6 week intervention phase (post-intervention assessment=12 weeks); 3 months after the end of the intervention phase (3-month follow-up assessment=6 months); and 9 months after completing the intervention phase (9-month follow-up assessment=12 months).

Most of the assessments were administered by a research assistant who was blind to whether people were in the treatment or control groups. Assessments of participants, who were asked not to disclose treatment status, included semi-structured interviews and questionnaires. A Rheumatology Nurse Specialist completed assessments of joint tenderness and arranged for blood tests to be carried out.

5.5 Measures

5.5.1 Selection of outcome measures

The International League of Associations for Rheumatology (ILAR) has been meeting with the World Health Organization (WHO) on a regular basis to discuss issues of mutual interest. Over the last few years these meetings have focused on the development of outcome measures in the rheumatic diseases, principally driven through OMERACT (Outcome Measures for Arthritis Clinical Trials). The Sixth Joint WHO/ILAR Task Force Meeting on Rheumatic Diseases reviewed a number of outcome measures for rheumatic diseases that had been developed over the past few years under the aegis of OMERACT (Brooks & Hochberg, 2001). The WHO/ILAR meeting formally endorsed these outcome measures and acknowledged them as the gold standard for outcome measures in these conditions

Outcome measures for rheumatoid arthritis clinical trials were developed at OMERACT 1 (Tugwell & Boers, 1993) and are very similar to those developed by the ACR (Felson *et al.*, 1993). The recommendations for the preliminary core set were:

- Acute-phase reactants
- Patient rating of physical disability
- Joint pain/tenderness
- Joint swelling
- Patient rating of pain
- Patient global assessment of disease activity

- Physician global assessment of disease activity
- Radiographs for studies of 1 yr or longer.

Together the core set samples the broad range of possible dimensions reflecting improvement in disease activity in RA, i.e. it has good content validity. OMERACT is continuing to develop this core set to include other dimensions of clinical outcome (e.g. psychological well-being).

Although this consensus was reached after the start of the current study, measures assessing many of the core set of outcomes for RA clinical trials were included. No measures of swollen joint count, physician global rating or radiographic changes were included. In this clinical trial the outcome domains that are likely to show change are short term and primarily though not exclusively patient centered. Measures were selected to sample three primary targets (end points) for the self-management intervention:

1. Pain;
2. Physical disability;
3. Emotional Distress

Measures of secondary targets were also included:

1. Social and occupational function;
2. Individualized quality of life;
3. Disease activity (Inflammation/Acute phase response; tender joint score);
4. Health care utilization.

Measures were also selected to sample the processes proposed in Leventhal's self-regulatory model which may mediate or moderate the effect of the self-management intervention on RA outcome. The measures were trialled in a pilot study (Pimm *et al.*, 1992) and certain measures were modified or rejected because of problems such as excessive length or limited responsiveness.

5.5.2 Demographic data

Demographic data collected included age, sex, marital status, number of children, ethnic origin, occupational status, and educational level.

5.5.3 Primary outcome measures

5.5.3(a) Pain

Pain is one of the core outcomes for RA clinical trials and is a primary end point for the intervention. Jensen (1992) reviews the use of pain rating scales suggesting that comprehensive assessment of pain experience should include intensity, distress, and location. Studies have shown that visual analogue scales have acceptable reliability and validity (Beery and Huskisson, 1972; Huskisson, 1974, 1983; Revill *et al.*, 1976). In RA visual analogue scales for pain have been found to have acceptable test re-test reliability and face, content, criterion and discriminant validity Felson *et al.*, 1993; McCarty 1974). U.S. studies of the Arthritis Self-Management Programme have used graphic rating scales for pain (GRSP). British intervention studies for arthritis have used visual analogue scales (VAS). Jensen (1992) reports that the NRS 101 (NRIP) is more easily completed by older people. Therefore to allow comparison with other studies and assess the relative properties of the scales, pain intensity was assessed using three different rating scales including: visual analogue scale (BVAS); graphic rating scale (GRSP) and numerical rating scale NRS 101 (NRIP). These measures involve selection of a point on a 10cm line that reflects the degree of pain intensity (see Appendix F for copies of scales). The distance (mm) from the no pain anchored end of the scale to the mark was measured using a ruler. High scores indicate worse pain. The AIMS2 Pain scale was also used to provide an alternative Likert type rating scale (see AIMS2 description below). Pain distress was assessed using a NRS 101 rating scale. The Ritchie Articular Index, discussed below, also provides a less subjective assessment of joint pain.

5.5.3(b) *Physical disability*

Physical disability is one of the core outcomes for RA clinical trials and it is a primary end point for the intervention. It was assessed using three different questionnaires.

The Stanford Health Assessment Questionnaire (HAQ) (Fries, Spitz, Kraines and Holman, 1980) is the most widely used measure of functional ability for people with arthritis. The HAQ comprises 20 activities of daily living questions, grouped into eight functional categories. Level of difficulty over the previous week is recorded (no difficulty, some difficulty, much difficulty, or unable to do, 0-3) and aids or assistance required. Within each of the eight functional categories the item with the highest score is used, and the score is adjusted to two when patients report the use of technical aids or require assistance from another person. The eight category scores are summed and averaged; yielding a disability score of 0-3, where three is extreme disability.

The HAQ has good test re-test reliability, content, face, criterion and discriminant validity (Felson *et al.*, 1993). It was used in the evaluation of the U.S. ASMP and should provide comparison data. The HAQ has been modified for use in British populations, by Kirwan and Reeback (1986), and this version was used in the present study. They found that the British version of the HAQ was acceptable to patients, reliably completed by patients as judged by rheumatologists interviews of patients and was more sensitive to change than the Steinbrocker Functional Class.

Physical disability was also measured using the *Arthritis Measurement Scales 2 (AIMS2)*. The AIMS2 is an improved version of the AIMS (Meenan, Mason, Anderson, Guccione & Kazis, 1992). The AIMS2 is a multidimensional measure of the health status component of patient outcome in the rheumatic diseases. It can be characterised as a disease specific health related quality of life measure. In addition to measuring physical disability the AIMS2 also measures other dimensions of health status including pain, emotional distress, social interaction and occupational function.

The AIMS 2 has 12 specific health status scales, summary components and overall impact items. There are 78 self-report items and health status is rated over the past two weeks. A normalization procedure is used so that all scale scores are expressed in the range 0 to 10, with 0 representing good health status and 10 representing poor health status. The scaling properties, reliability and validity of the AIMS were reported by Meenan, Gertman & Mason (1980) and Meenan, Gertman, Mason and Dunaif (1982). The AIMS 2 has also been shown to have satisfactory internal consistency, test re-test reliability, and internal validity (Meenan *et al.*, 1992). The AIMS has been shown to be sensitive to clinical improvement in patients with RA and OA (Anderson, Firschein and Meenan 1989; Meenan *et al.*, 1984).

Factor analysis has shown that the 12 original AIMS scales can be combined into three or five component models of health status (Mason, Anderson & Meenan, 1988). The 12 AIMS2 scales can be grouped and scored to generate five components of health status: Physical (Mobility, Walking and Bending, Hand and Finger Function, Arm Function, Self Care and Household Tasks); Affect (level of Tension and Mood); Symptoms (Arthritis Pain); Social Interaction (Social Activity and Support From Family) Role (Work) (Meenan *et al.*, 1992).

Performance of everyday activities for arthritis was assessed on a questionnaire developed to evaluate changes in function following the ASMP, the Physical Activities/Therapies for Arthritis scale (Holman and Lorig, 1992). Participants rate how often they perform each of 18 everyday activities on a 6-point rating scale from never to very often. The activities on the scale are grouped into four sub-scales, chores (household tasks), Work (gardening and DIY), Social (seeing friends and relatives) and Outings (going out e.g. to a park, for a meal or movie). The psychometric properties of the scale have not been reported.

5.5.3(c) *Emotional state*

Emotional distress is common in RA and it was a primary end point for the intervention.

Anxiety and depression were assessed using the Hospital Anxiety and Depression Scales (HADS) Zigmond and Snaith (1983). The HADS was developed for use with medical outpatients but is now widely used in clinical practice and research (Herrmann, 1997). It is a 14 item self-report measure of symptoms of anxiety and depression over the past week. There are seven anxiety and seven depression items each scored on a 0-3 scale. Interpretation is usually based on cut off scores and Snaith and Zigmond (1994) recommend that for both anxiety and depression scales a raw score of 8-10 indicates mild cases, 11-15 moderate cases and 16-21 severe cases. A raw score of 8-10 indicates a possible case and 11 or above indicates a definite case. These cut offs, in addition to mean scores, are used in the present study.

Crawford *et al.*, (2001) has recently reported normative data for the general adult population in Great Britain. This allows comparison between the means and standard deviations for clinical and general population samples. They found that females had significantly higher means for both anxiety and depression scales so interpretation should be gender specific.

The HADS has good internal consistency (Moorey *et al.*, 1991) and satisfactory reliability and validity (Clark and Fallowfield, 1986), good face and construct validity (Moorey *et al.*, 1991) and concurrent validity Zigmond and Snaith (1983). The HADS has been used successfully to evaluate the efficacy of interventions for people with arthritis in the UK (Barlow, Wright and Kroll, 2001).

People with RA may experience a wider range of emotional reactions than anxiety or depression (e.g. frustration or anger). Further positive mood has been shown to be a dimension of emotional state which is independent of negative mood. A study of RA patients has shown that although positive and negative affect are negatively correlated, they constitute two separate and distinct emotional responses (Zautra *et al.*, 1995). Therefore a wider range of assessments of emotional state were used including AIMS2 Affect (see above), the Profile of Mood States [POMS] (McNair, Lorr and Droppleman 1992) and the Positive and Negative Affect Scale (PANAS) (Watson, Clark and Tellegen, 1988).

The POMS is a 65-item mood adjective checklist with six sub-scales: tension/anxiety, depression/rejection, anger/hostility, vigour/activity, fatigue/inertia and confusion/bewilderment. Participants rate the extent to which they have experienced each specific emotion over the past week on a 0 (not at all) to 4 (extremely) scale. The POMS has been shown to have satisfactory internal consistency, test re-test reliability and face, predictive and construct validity as a measure of mood states in psychiatric and general adult populations. It is sensitive to change in therapy (McNair, Lorr and Droppleman, 1992)

The PANAS is a mood adjective checklist with positive and negative affect scales (Watson *et al.*, 1988). It has 20 items 10 positive and 10 negative. Both scales are scored on a five-point rating scale from not at all to extremely. The state version of the PANAS was used in this study. The instructions used asked participants to consider their mood over the past week. Watson *et al.*, (1988) reported the psychometric properties of the PANAS. Both subscales have satisfactory internal consistency and test re-test reliability. The two sub scales were found to be independent and this was confirmed using factor analysis. PANAS also had acceptable concurrent validity as it was significantly correlated with other measures of distress and psychopathology. The PANAS has been used to evaluate interventions for people with arthritis (Barlow *et al.*, 2001).

5.5.4 Secondary outcomes

5.5.4(a) Social and occupational function

These were measured using the AIMS2 Social interaction and Role scales (see above).

5.5.4(b) Individualized quality of life (PGI)

Most measures of health related quality of life (HRQoL) do not take into account the

person's judgements of the relative importance of the different areas of their lives affected by the disease (Carr and Thompson, 1994) Therefore an individualized measure of the impact of arthritis on the person's quality of life (HRQoL), the modified Patient Generated Index (Ruta, Garratt, Leng, Russell and MacDonald, 1994) was included. The PGI measures people's views about the specific impact of a health problem on their quality of life and their priorities for improvement. Unlike other generic or disease specific measures of HRQoL the PGI allows people the freedom to choose any areas of life that they feel are important rather than responding to a pre-determined list. Participants completing the PGI:

1. Identify up to five areas of life affected by RA;
2. Rate the severity of impact on each area on a rating scale from 0 the worst you can imagine to 10 exactly as you would like to be;
3. Indicate priorities for improvement by allocating 12 spending points between the areas.

This allows the calculation of an index score from 0-10, with higher index scores indicating higher individualized HRQoL. To measure change, on subsequent assessments participants were given the PGI questionnaire with the areas of life and spending points they had identified at the first assessment already inserted. They were asked to rate the current severity of impact for each area. This procedure was developed for the present study to ensure that change on the PGI reflected change in the impact of RA on areas of life that the participant had identified as being affected by RA rather than reflecting a shift in focus to new areas or priorities. The measure has acceptable reliability and validity (Ruta *et al.*, 1994).

The PGI has been found to be a useful measure of HRQoL in rheumatological conditions e.g. chronic pain (Ruta *et al.*, 1994), RA (Pimm *et al.*, 1997) and Ankylosing Spondylitis (AS; Haywood, Garratt, Dziedzic & Dawes, 2003). The PGI has also been used in other health conditions, e.g. heart disease (Smith, Taylor & Mitchell, 2000), atopic dermatitis (Herd, Tidman, Ruta & Hunter, 1997) and sleep apnoea (Jenkinson, Stradling & Petersen, 1998). Dempster, Donnelly and Fitzsimons (2002) found that,

when compared to generic and disease specific HRQoL measures, the PGI measured unique features of HRQoL. They also report that the PGI had acceptable reliability and validity when compared to generic and disease specific HRQoL measures, although sensitivity to change was not demonstrated.

5.5.4 (c) Disease activity

Felson *et al.*, (1993) reviewed outcome measures for disease activity in RA and they found that the majority of the commonly used measures (e.g. Ritchie Articular Index; ESR; CRP) have at least moderate sensitivity to change. Many of the measures including: EMS, ESR and tender/swollen joint counts have good criterion validity as they predict important long-term outcomes in RA (e.g. mortality; disability; radiographic damage). The measures also have good test re-test reliability and as mentioned above content validity.

Rheumatoid Factor was only measured at baseline. Blood tests carried out at baseline and both follow ups included: ESR; CRP and full blood count (haemoglobin, platelets and white cell count). To monitor the acute phase response over the course of the study, CRP was also measured at the end of the six-week baseline and following the intervention. ESR and CRP are the two most widely used laboratory tests for inflammation (acute phase reactants) in RA. ESR and CRP are highly correlated and so it was not necessary to give both at all time points. For people attending the hospital outpatient clinic ESR was routinely measured every six months. However in people with long standing RA over 20 years duration the ESR may be persistently elevated with no clinical evidence of inflammation. Therefore the CRP was chosen as the primary measure of inflammation as it is likely to be more sensitive to change in this sample.

The duration of early morning stiffness (EMS), recorded in minutes, was obtained in a standardized manner (McCarty, 1974). EMS is not very sensitive to change because of the variability of responses. However it is an important component of the person's experience of RA and it is predictive of important long-term disease outcomes and so it was included in the study.

Joint tenderness was evaluated at all assessments using the Ritchie Articular Index. This is a well-established clinical measure of joint tenderness (Ritchie *et al.*, 1968). A trained Rheumatology Nurse Specialist graded 52 peripheral joints, or joint areas, on a four-point rating scale, for tenderness. To reduce inter-observer variability the assessments for each participant were carried out by the same assessor. It has been shown to be one of the most responsive and sensitive to change of the outcome measures used in RA clinical trials (Dixon. Hayes, Constable & Bird, 1988)

5.5.4(d) Health care utilization

The use of health service resources was assessed by self-report. Participants were asked to report the frequency of medical consultations for their RA over the past four months. Similar self-report data on the use of health service resources was collected in a four-year follow up study of the US ASMP. This measure was sensitive to the reduction in medical consultations following the intervention (Holman and Lorig, 1992).

5.5.5 Self-regulatory variables

Assessments of psychological variables that might predict, moderate or mediate the relationship between the intervention and outcome included: illness representations, coping strategies, self-management behaviours and perceived self-efficacy.

5.5.5(a) Illness representations

Illness representations were assessed using the Illness Perception Questionnaire. The IPQ was developed to provide a quantitative assessment of the five cognitive components of the illness representation (identity, consequences, timeline, control/cure and cause) in Leventhal's Self-Regulatory Model (Leventhal *et al.*, 1980, 1984, 1992a). A small cohort of people with RA were included in the original studies developing the IPQ (Weinman *et al.*, 1996). The first version of the IPQ was used in this study. It has since undergone further development (Weinman *et al.*, 1996) and

revision (Moss-Morris *et al.*, 2002). It has been used in studies of illness adaptation in patients with a wide range of conditions, including heart disease (Cooper, Lloyd, Weinman & Jackson, 1999; Petrie, Weinman, Sharpe & Buckey, 1996; Steed, Newman & Hardman, 1999), rheumatoid arthritis (Murphy *et al.*, 1999; Pimm & Weinman, 1998; Scharloo *et al.*, 1999), cancer (Buick, 1997; Buick and Petrie, 2002), psoriasis (Fortune, Richards, Main & Griffiths, 2000), Scharloo, Kaptein, Weinman, Vermeer & Rooijmans, 2000a), chronic obstructive pulmonary disease (Scharloo *et al.*, 2000b), chronic fatigue syndrome (Heijmans, 1998; Moss-Morris, Petrie & Weinman, 1996), diabetes (Griva, Myers & Newman, 2000) and Addisons disease (Heijmans, 1999). It has also been adapted for use with people undergoing investigations such as coronary angiography and genetic testing, and for spouses and carers of people with major health problems (Heijmans, de Riddler & Bensing, 1999; McClenahan & Weinman, 1998; Weinman, Petrie, Sharpe & Walker, 2000).

This study used the original IPQ. It has forty items and six sub-scales assessing different components of the illness representation. The sub-scales are: Illness identity (perceptions of the range and frequency of RA symptoms; twelve items), Symptom Coherence (perceptions of how puzzling, distressing and changeable RA symptoms are three items), Time Line (perceived chronicity of RA; three items), Consequences (perceptions of the seriousness of the consequences of RA; four items), Control/Cure (perceptions of the controllability/curability of RA five items) and Cause (the extent to which people perceive specific factors to have caused their RA eight items). On the Identity scale of the original IPQ participants record how frequently they have experienced each of 12 symptoms, as part of their arthritis, on a 4 point scale 0 never to 3 all the time). The other twenty-eight items are rated on a 5 point scale from 1 strongly disagree to 5 strongly agree. For the IPQ sub-scales high scores indicate that RA is perceived as: having a stronger illness identity, greater range and frequency of symptoms attributed to the illness (Illness Identity); less coherent (Symptom Coherence); more chronic (Timeline); having more serious consequences (Consequences) and being more controllable (Control/Cure). Previous research has suggested that it is not appropriate to combine the eight specific cause items into a single scale as they assess disparate constructs (Weinman *et al.*, 1996). It is recommended that the cause items are analysed

as single items. However, previous research has tended to omit the cause items from the analyses because of statistical problems with including a large number of single item variables (Scharloo *et al.*, 1999). In the present study an attempt was made to construct a single scale reflecting the number of causes people identified for their RA. This was constructed by recoding responses to cause items as either 1=cause (agree or strongly agree) or 0=not a cause (neither agree nor disagree, disagree or strongly disagree). The eight items were then summed to produce a single scale with a 0-8 range.

Weinman *et al.*, (1996) and Moss-Morris *et al.*, (2002) have reported on the psychometric properties of the IPQ and IPQ-r. Factor analysis has confirmed the factor structure of the IPQ-r. The scales have satisfactory internal consistency and test re-test reliability, and sound discriminant, known group and predictive validity have also been demonstrated.

5.5.5(b) *Self-efficacy*

Self-efficacy has been shown to be an important predictor of outcome of the ASMP and Holman and Lorig (1992) suggest that the course outcomes are mediated by changes in self-efficacy. Self-efficacy was assessed using the Arthritis Self-Efficacy (ASE) scale developed to measure self-efficacy in people with arthritis (Lorig *et al.*, 1989a). This is a 20-item questionnaire with three subscales measuring the person's confidence in their ability to manage arthritis pain (five items), other arthritis symptoms (six items) and functional activities affected by arthritis (nine items). For each of the items participants rate their degree of confidence on a 10-point rating scale ranging from 10 (Very uncertain) to 100 (very certain). Scores are summed in each scale and higher scores indicate higher self-efficacy. Lorig *et al.*, (1989a) report that for US samples of people with arthritis the ASE scale has satisfactory internal consistency, test-retest reliability, and construct and concurrent validity. The factor structure was supported by confirmatory factor analysis.

The ASE scale has been shown to be sensitive to change over the course of the ASMP Holman and Lorig (1992). The comprehensibility, reliability and validity of the ASE

scale have been investigated in 4 studies of community-based Arthritis Self-Management Programmes in the UK (Barlow, Williams & Wright, 1997c). They concluded that the ASE is a reliable and valid measure of arthritis self-efficacy for use among community-based samples of people with arthritis in the UK and may be a useful indicator of change in evaluations of arthritis self-management courses.

The Generalized Self-Efficacy Scale (Jerusalem & Schwarzer, 1992) was also administered. This assesses the degree to which individuals feel confident in their ability to respond to demanding situations or events and has been validated for use with people with arthritis in the UK (Barlow *et al.*, 1996) and has been used to evaluate interventions for people with arthritis (Barlow *et al.*, 2001). Participants scores are on a four point rating scale (1-4) ranging from 'Not at all' to 'Exactly true. Scores are summed across the ten items. Higher scores indicate higher levels of self-efficacy.

5.5.5(c) Coping procedures

Coping procedures were sampled using a number of different approaches. These included a generic coping strategies questionnaire (COPE), a pain specific coping strategy questionnaire (Coping Strategy Questionnaire) and a questionnaire measuring arthritis specific self- management behaviours (Physical Activities /Therapies For Arthritis).

Generic coping strategies were assessed using a modified version of the COPE (Carver, Scheier & Weintraub 1989). The COPE is a self-report instrument with 60 items rated on a 4-point scale (1 - I usually don't do this at all to 4 - I usually do this a lot). The COPE has 15 conceptually distinct scales assessing adaptive and maladaptive aspects of coping. The COPE has been used in a wide variety of illness groups. It has been shown to be a valid and reliable measure of coping. It has satisfactory internal consistency, test re-test reliability, construct [convergent and discriminant] validity. Factor analysis of 13 subscales confirms 11 factors. The two social support sub scales loaded together as did the planning and active coping sub scales. In this study a short version of the COPE was

used which was initially developed by Carver *et al.*, (1993) and was further modified following the pilot study (Pimm *et al.*, 1992) to reduce the number of items further. Items that reduce the internal consistency of a subscale or that had the lowest correlation coefficient with the subscale were dropped. The situational version of the COPE scale was used with participants rating what they had done over the past two weeks. The results of the pilot study indicated the Turning to religion and Alcohol/Drug use subscales had little relationship to the outcome measures and so they were not included. This created a 26-item scale with 13 sub scales. Carver (1999) has reported that a very similar short version of the COPE has good psychometric properties.

The Pain Coping Strategies questionnaire (CSQ; Rosenstiel & Keefe, 1983) is a 44-item questionnaire which assesses the use of coping strategies for pain and the extent to which the strategies are perceived as effective in the control or reduction of pain. There are six different cognitive coping subscales (six items), one behavioural coping scale (six items) and two items assessing pain control and efficacy beliefs. All items use a seven-point Likert scale for responses. A shorter version of the scale which was used to evaluate the US ASMP was used in this study. This did not include the praying and hoping and catastrophizing subscales or the two pain control belief questions, and so it had five subscales and 30 items.

Internal consistency of the CSQ subscales is satisfactory. Rosenstiel and Keefe (1983) identified three factors in factor analysis: cognitive coping and suppression (reinterpreting coping self statements and ignoring); helplessness (catastrophizing, increasing activity, control and ability to decrease pain); Diverting attention and praying/hoping. Evidence on the validity of the three factor scores comes from a multiple regression analysis predicting five different adjustment measurements (Rosenstiel & Keefe, 1983). People scoring high on factor one were more likely to report having functional impairment (the version of the CSQ used in this study contained all the subscales making up this factor), those scoring high on factor two scored high on anxiety and depression measures, those scoring high on factor three had higher pain and functional impairment. There was some evidence of concurrent validity, Crisson and Keefe (1988) found positive correlations between beliefs on the

Multidimensional Health Locus of Control Scales and factors two and three of the CSQ.

Practice of arthritis self-management behaviours was assessed on items from the Physical Activities/Therapies For Arthritis questionnaire (see above). Items were designed to measure frequency and duration of practice of a number of arthritis specific self-management behaviours (Holman & Lorig, 1992). There are seven frequency items on which people report how many times a week they have practised seven self-management behaviours (stretching exercises, strengthening exercises, relaxation, massage, walking over 1/2 mile, swimming and bicycling) over the past fortnight. There are four duration items on which patients report how long they exercised for (minutes walking, swimming, bicycling and miles walked). These questions have been shown to be sensitive to change over the course of the Arthritis Self-Management Programme (Holman & Lorig 1992).

5.6 Detailed procedure

1. Participants were recruited from the Monday and Tuesday afternoon Rheumatology outpatient clinics at Stoke Mandeville Hospital.
2. All potential participants who satisfied the inclusion and exclusion criteria and who were due to attend the out-patient clinic were identified by the Rheumatology Nurse Specialist or research assistant from available medical notes.
3. The Rheumatology Nurse Specialist or research assistant gave all potential participants attending the out-patient clinic the study information sheet and patient informed consent form to complete before their appointment with the clinic doctor. The patient's name and address stickers and their telephone number were put on the informed consent forms and a medical consent form was attached to the medical notes. A sticker was attached to the medical notes of all people who were invited to participate in the study to ensure that people were not re-invited.
4. If the patient and the clinic doctor were happy for the patient to participate in the study the doctor signed the medical consent form. If the doctor was not happy they completed the medical consent form giving reasons for exclusion from the study. The doctor gave

the patient the medical and patient informed consent forms. The Rheumatology Nurse Specialist or research assistant collected patient and medical informed consent forms from the patients.

5. At the point of recruitment standard medical assessments of disease activity and severity were carried out on all participants including early morning stiffness, Ritchie Articular Index, full blood count, ESR, CRP, and Rheumatoid Factor. Basic information was collected for all participants who did not wish to participate in the study. This included: age, sex, diagnosis, duration and distance from the research center. This was collected to allow comparison of participants who agreed/did not agree to participate in the study. The Rheumatology Nurse Specialist ensured this data was collected at the clinic. The GP was informed by letter that the participant was participating in the study.
6. Participants were recruited in batches, of 20, six to eight weeks before each Arthritis Self-Management Course. Throughout the study the time of each participant's appointments was kept constant to minimize the effects of diurnal changes on outcome measures (e.g. pain). Details of participants who were willing to participate but who were prevented at present (e.g. transport difficulties or work) were kept. When sufficient participants were recruited for these special batches, (e.g. working people), they were passed for randomization.
7. Details of participants who agreed to participate were passed to the principal researcher for randomization. Participants were randomly allocated to treatment and control groups. Participants in the treatment group were invited to participate in the Arthritis Self-Management Course. They were asked not to inform the research assistant or other people who may be participating in the study that they were attending the course.
8. Details of all participants who agreed to participate in the study were passed to the research assistant. They then arranged to interview them and complete the standardized questionnaires.
9. In the fortnight preceding the Arthritis Self-Management Course all participants were assessed by the Rheumatology Nurse Specialist at an outpatient clinic. She completed a Ritchie Articular Index and arranged for a CRP blood test.
10. In the fortnight preceding the Arthritis Self-Management Course the research assistant repeated the questionnaires with all participants. Questionnaires were posted to participants one week before their assessment and they were asked to complete them

before meeting the Research Assistant. At the assessment the Research Assistant scrutinized the questionnaires, answered any questions and filled in the questionnaires for any participants who were unable to write.

11. Participants who agreed to attend the Arthritis Self-Management Course then attended the six weekly group sessions which were led by the Principal Researcher and Rheumatology Nurse Specialist. A record of attendance was kept.
12. In the fortnight following the course all participants were assessed by the Rheumatology Nurse Specialist who completed a Ritchie Articular Index, arrange for a CRP, and for participants in the treatment group to complete the post-treatment ratings.
13. In the fortnight following the course the research assistant repeated the questionnaires with all participants following the procedures in 10 (above).
14. 12-13 weeks after completion of the Arthritis Self-Management Course all participants were re-assessed in the Rheumatology outpatient clinic. Standard medical assessments of disease activity and severity were completed.
15. 12-13 weeks after completion of the Arthritis Self-Management Course the research assistant completed the follow-up questionnaires with all participants following the procedures in 10 (above).
16. Six months after the first follow-up assessment the participants were re-assessed in the Rheumatology outpatient clinic. Standard medical assessments of disease activity and severity were completed.
17. Six months after the first follow-up assessment the participants were re-assessed by the research assistant who repeated the questionnaires following the procedures in 10 (above)

18. All participants who participated in the research were debriefed after their final assessment session. For participants in the control group explanation of the full nature and purpose of the study was particularly careful. Participants in the control group were eligible to participate in the self-management programmes run after completion of the study.

The data collection phase of the study began in 1994 and took three years to complete. The bulk of the interventions were conducted during the first two years. The third year was devoted to follow up assessments.

5.7 The Self-management intervention

The intervention was developed in a pilot study (Pimm *et al.*, 1992). The pilot intervention was based on the Arthritis Self-Management Programme (Lorig *et al.*, 1989a) and Tucker & Kirwan (1991) in-patient education programme. The intervention used in this study was based on the revised US Arthritis Self-Management (Help) Course (Lorig and Fries 1990) adapted for the UK, people with RA and in the light of experience from the pilot study. Course leaders used the Arthritis Self-Management Course Leaders manual and participants used the Arthritis Help Book (Lorig and Fries 1990). Both publications provide further information about the content of the self-management intervention. The treatment manual is included in Appendix H.

Participants in the intervention groups attended six weekly two-hour meetings. The groups were led by the Principal Researcher (Clinical Psychologist) and Rheumatology Nurse Specialist assisted by trained people with arthritis. Input to specific sessions was provided by other members of the multidisciplinary hospital team including occupational therapists, physiotherapists, podiatrists and social workers.

The programme had explicit objectives. The format of the groups was interactive, participants being encouraged to share their perceptions of RA and coping procedures which had been helpful. A range of cognitive and behavioural strategies were used.

Information about their disease and techniques for coping with it were taught. Participants were encouraged to work on specific goals and monitor their progress between sessions.

The sessions covered the following information and coping skills: the nature of RA, medication, joint protection, use of aids, exercise, hot and cold therapy, foot care, nutrition, psychological adjustment (e.g. coping with depression), coping with illness (e.g. problem solving and communication skills), stress and pain management (e.g. distraction, visual imagery, relaxation), social relationships, occupation, leisure, and finance (e.g. use of community resources).

Each self-management group had 6-8 participants and so 7-9 self-management groups were planned and eight groups were run.

5.8 Ethical considerations

Ethical approval for the study was sought and obtained from the participating hospital's ethics committee (see Appendix B).

The main ethical issues considered in planning the study were the following:

5.8.1 Confidentiality

Participants' files and Questionnaires were identified only by a numerical code, in order to ensure efficient administration of data collection. Therefore data was not fully anonymous. All records were kept confidentially in accordance with Data Protection procedures.

5.8.2 Informed consent

All participants were given an information sheet about the study and a consent form to complete (Appendix E). The information sheet explained the background to the study, what participation in the study would entail and assured confidentiality of data. It stressed that participation was voluntary, that they were free to withdraw from the study at any time and their participation would not affect their normal hospital treatment. If they decided to participate, consent was also sought from their hospital doctor. Their general practitioner was informed about their participation in the study. Participants were then sent a questionnaire pack and contacted to invite them to an appointment at the hospital to carry out further assessments. This gave participants ample time to consider whether or not they wished to participate in the study.

The Zelen design raises some ethical issues, participants in the control group were aware that they were participating in a study of how people cope with RA but were not aware that a different group of participants were receiving a treatment. It is possible that participants in such a study might feel distressed that they were not fully informed about the study's purpose. Participants in the control group were carefully debriefed at the end of the study and were offered the treatment (Appendix G). Participants in the treatment group were initially recruited into a study about how people cope with RA and following full discussion of the study's aims they were then offered the treatment. Participants were free to decline the treatment and to continue in the study or withdraw. No participants expressed any concerns about the study.

5.8.3 Adverse effects

Participants had to complete a series of questionnaires, a joint examination and blood tests. These procedures may cause minor discomfort and distress but did not greatly exceed the demands made by routine care, e.g. people with RA frequently have monthly blood tests as part of the normal management of their RA. Participants in the treatment group were invited to attend the self-management intervention. The intervention had

been extensively investigated in the USA and no adverse effects had been reported.

If the researchers identified at any point during the study that any participant was experiencing a significant physical, psychological or social difficulty this was recorded and they were immediately referred to the appropriate service.

5.8.4 Withholding intervention

Randomized controlled trials can raise important ethical issues. In this study participants in the control group did not receive the treatment for one year. The control group did receive standard care and so were no worse off than if they had not participated. The treatment had not been evaluated in the UK and so the potential benefits and costs were unclear. Capacity to deliver the treatment was limited. Finally all participants in the control group were offered the treatment at the end of the study.

5.8.5 Debriefing

All participants were fully debriefed after their final assessment (Appendix G). Following completion of the data collection phase of the study all participants were also invited to feedback meetings where the preliminary results were discussed.

Chapter 6 Results: Overview, sample characteristics and representativeness

6.1 Overview of results

The results are divided into four chapters. Chapter 6 presents the sample characteristics and representativeness. This includes descriptive data and statistical analyses of response rates and the sample's demographic data and baseline scores for the main response variables. This chapter also addresses Research Questions 1.1, 1.2 and 1.3. Chapter 7 presents the results of the statistical analyses exploring Research Questions 1.4 and 1.5 and testing Hypothesis 1.1 and 1.2. Chapter 8 presents the results of statistical analyses testing Hypothesis 2.1-4 and Chapter 9 presents the results of analyses testing Hypothesis 3.1 and 3.2.

The Statistical Package for Social Sciences for Windows (SPSS) was used to conduct the statistical analyses. A detailed description of the statistical methods used is provided at the start of each of the chapters.

Scores from the questionnaires can be treated as interval level variables for the purpose of statistical analysis (Fife-Schaw, 2000; Labovitz, 1971). In addition, the central limit theorem enables us to assume an underlying normal sampling distribution, due to the large sample size (Aron & Aron, 1999) and the Kolmogorov- Smirnov test ensured that subgroups with small numbers had approximately normal distributions. Therefore, parametric tests were used except where the assumptions for parametric tests were violated.

6.2 Statistical methods - sample characteristics and representativeness

Pearson's Chi square, Yate's Continuity Correction, two tailed independent samples T-tests and Mann Whitney u tests were used to test for differences between:

1. participants and non-participants,
2. participants who provided data and those who provided no data,
3. participants who completed the study and those who provided no data or withdrew,
4. participants who completed the study and those who withdrew during the study,
5. Participants in the treatment group who completed treatment and those that did not,
6. Participants in the treatment and control groups.

For the Chi Square tests where the expected values for one or more cells was less than five Yate's Continuity Correction was used. For the independent samples t-tests the data was assessed to check whether it met the assumptions for the use of parametric statistics. The majority of questionnaire measures used equal interval scaling and consisted of subscales of at least four items, so provided a spread of data distribution. The homogeneity of variance assumption was tested using Levene's test to check whether the variances of the samples were significantly different. If Levene's test was significant then the t-test assuming non-equal variances was used. Kolmogorov-Smirnoff one-sample tests were used to test whether the sample's scores were normally distributed. The assumption that the population from which the sample was drawn is normally distributed was not met if Z was significant at the $p=0.05$ level. The results of the Levene's test and Kolmogorov-Smirnoff Tests are only reported if they are significant. As it is not possible to be completely certain that the assumptions for parametric tests have been met non-parametric Mann-Witney U tests were also performed. The results of non-parametric tests are only reported if they differ significantly from the results of the parametric tests. The number of statistical tests conducted on the same data set increases the risk of a type 1 error, where appropriate a Bonferroni correction is made.

Descriptive data for the main response variables at baseline was compared to normative data for the measures to answer Research Question 1.1.

6.3 Eligibility

The study had explicit inclusion and exclusion criteria. Table 6.1 shows the number of participants who met/did not meet each inclusion and exclusion criterion. People who were entered into the study who met the inclusion and exclusion criteria are referred to as “eligible” participants. Those who did not meet the criteria will be referred to as “ineligible participants”. Some people who met the criteria when they entered the study later developed a problem, which would have made them ineligible had it been present at study entry. These participants are referred to as “late ineligible participants”.

Table 6.1: Number of ineligible participants in the sample for each inclusion and exclusion criterion.

Eligibility Criteria	Eligible	Ineligible
American Rheumatism Association Revised diagnostic criteria met	140	0
More than one year since diagnosis	140	0
Within age range 16-70	140	0
Ability to attend required number of hospital appointments & group sessions	140	0
Reasonable understanding of English	136	4
No history of severe mental illness	140	0
Total	136 (97.1%)	4 (2.9%)

Considering each inclusion and exclusion criterion:

1. American Rheumatism Association 1987 revised diagnostic criteria for rheumatoid *arthritis*.

All participants in the study met this criterion at study entry.

There were two late ineligibles, both in the intervention group, whose diagnosis changed during the study. Participant 16 began investigations for systemic lupus erythematosus and dropped out of the study before the first assessment. Participant 113 was given a diagnosis of cryoglobulinemia after the fourth assessment and died at this point. Their data has been included in the analysis as they met the criteria at study entry.

2. More than one year since diagnosis.

All participants in the sample met this criterion. The range of time since diagnosis based on self-report data was 1-47 years. The range of time since diagnosis, based on Data collected from medical notes was 1-35 years.

3. Aged 16-70 years.

All participants met this criterion, the age range for the study was 22-69 years.

4. Able to attend five hospital appointments and six weekly group sessions over the next year.

All participants reported that they were able to do this but as can be seen from Table 6.10 several participants gave reasons of time, distance, and transport for withdrawing from the study.

5. Reasonable understanding of English.

Four people entered in the study were found to have difficulty completing questionnaires because of poor understanding of written English. Two of these were in the treatment condition, participants 30 and 69, and two in the control condition, participants 88 and 111. These participants were considered to be ineligible at study entry and their data was not included in the analyses. Formal screening of ability to understand written English was not included in the selection procedure. This may explain why these ineligible participants were entered in the study.

6. No history of severe mental illness:

No participants were identified as having a history of severe mental illness at study entry or during the study. During the study two participants were found to be experiencing mild/moderate mental health problems. Participant 10 who was in the intervention condition was suffering from anxiety, and participant 57 who was in the control condition was suffering from anxiety and depression. Following the study protocol that participants should receive standard medical care they were referred for individual psychological therapy and subsequently dropped out of the study. As these participants were considered to meet the criteria at study entry their data was included in the analyses.

6.4 Response rates and comparison of participants and non-participants

The sample was recruited in batches from consecutive attenders at the hospital outpatient clinic. Using medical notes 400 people were identified as meeting the inclusion and exclusion criteria prior to their appointment. Of these 136 (34%) agreed to participate in the study.

It is important to determine whether the study sample is representative of the population from which it is drawn. Statistical analyses were computed to test for differences between eligible participants and non-participants on sex, age, duration and distance. Table 6.2

presents data for study participants and people who were eligible for the study but did not participate.

Table 6.2: Comparison of sex, age, duration of illness and distance from research centre for study participants and people who did not participate.

	Non-participants	Participants	Statistical Test
N (% total)	264 (66%)	136 (34%)	
Sex	M = 72 (27.3%) F = 192 (72.7%)	M = 33 (24.3%) F = 103 (75.7%)	X ² = .420; df = 1; p = .517
Age	Mean 55.11 Range 18-70 s.d. 10.22 se 0.6289	Mean 53.55 Range 22-69 s.d. 10.03 se 0.8597	T = 1.57; df = 398; p = 0.117
Duration of Illness	Mean 11.65 Range 1-42 s.d. 9.31 se 0.6613	Mean 11.91 Range 1-35 s.d. 9.02 se 0.7761	T = -.26; df = 331; p = 0.797
Distance from home to centre	<15 miles = 126 47.7% >15 miles = 138 52.3% Median = 15-20 miles Mode = 20-25 miles	<15 miles = 87 64.0% >15 miles = 49 36.0% Median = 10-15 miles Mode = 0-5 miles	X ² = 9.513; df = 1; p = .002**

The statistical analyses indicate that people participating in this study were not significantly different from non-participants in sex, age or duration of RA but were significantly more likely to live nearer the research centre. This is not surprising as people attending the Rheumatology outpatient clinic live in a rural area and as the study required several visits to the hospital those living nearer would be more likely to participate. This evidence suggests the sample is representative of the population eligible to participate in the study. Table 6.3 presents non-participants reasons for not participating in the study. Transport, distance, time and work commitments were the main reasons for non-participation. This is understandable as the Rheumatology outpatient clinic serves a rural population and many people are likely to experience travel difficulties as a result of their RA.

Table 6.3: Reasons given for not participating in the study.

Reason given for non-consent	Frequency (%)
Transport	57 (21.59%)
No reasons given	54 (20.45%)
Distance	42 (15.91%)
DNA clinic appointment	27 (10.23%)
Working	24 (9.09%)
Time	21 (7.95%)
Not interested	15 (5.68%)
Missed	8 (3.03%)
Involved in previous study	4 (1.52%)
Inconvenience	4 (1.52%)
Other	8 (3.03%)
Total	264 (100%)

6.5 Comparison of participants who provided data with those who provided no data

A total of 136 people completed a consent form saying they were willing to participate in the study. Of these 118 attended their first assessment and returned their questionnaires a response rate of 86.8%. 18 (13.2%) did not provide any data.

Statistical analyses were computed to test for differences between participants who provided no data and those that provided data with respect to experimental condition, sex, age, duration of illness and distance from research centre. The results of the statistical analyses are presented in Table 6.4. In summary there were no differences between the group who provided data and those who provided no data in experimental condition, sex or distance from research centre. The group who provided no data were significantly older and had had RA longer.

Table 6.4: Comparison of sex, age, duration of illness, distance from research centre and experimental condition for participants who provided no data and those that provided data.

	Provided no data	Provided data	Statistical Test
N (% total)	18 (13.24%)	118 (86.76%)	
Sex	M = 6 (33.3%) F = 12 (66.6%)	M = 27(22.9%) F = 91 (77.1%)	X ² =0.928; df=1; p=0.335; Yates=0.447; df=1; p=0.504
Age	Mean 58.3 Range 48 – 67 s.d. 8.72 s.e.1.72	Mean 52.89 Range22 – 69 s.d.10.21 s.e. .94	T = 2.174; df = 134; p=.031;
Duration of illness	Mean 17.94 Range 1-34 s.d. 8.72 s.e. 2.1	Mean 11.05 Range1 – 35 s.d.8.76 s.e. .82	T = 2.944; df = 127; p = .004
Distance from research centre	<15 = 11 61.1% >15 = 7 38.9% Median =12.5 Miles Mode = 5-10 Miles	<15 = 75 63.6% >15 = 43 36.4% Median = 15 miles Mode = 5–10 miles	X ² = .040; df = 1; p = .841
Exp Condition	Treatment = 9 (50.0%) Control = 9 (50.0%)	Treatment = 67 (56.8%) Control = 51 (43.2%)	X ² = .291; df = 1; p = .589

Table 6.5: Reasons for providing no data given by the 18 participants who provided no data.

Reason	Treatment N (%)	Control N (%)	Total sample N (%)
DNA	1 (5.6%)	3 (16.7%)	4 (22.2%)
Personal	0	3 (16.7%)	3 (16.7%)
Health	1 (5.6%)	1 (5.6%)	2 (11.1%)
Time	2 (11.1%)	0	2 (11.1%)
Transport	1 (5.6%)	0	1 (5.6%)
Distance	1 (5.6%)	0	1 (5.6%)
Family	0	1 (5.6%)	1 (5.6%)
No reason	1 (5.6%)	0	1 (5.6%)
Psychological	1 (5.6%)	0	1 (5.6%)
Questionnaire	0	1 (5.6%)	1 (5.6%)
Diagnosis ¹	1 (5.6%)	0	1 (5.6%)
Total (N)	9 (50.0%)	9 (50.0%)	18

¹Participant 16 provided no data giving diagnosis as a reason as he began investigations for SLE as mentioned above.

As can be seen from Table 6.5 people who provided no data gave a variety of reasons including changes in their physical or psychological health, personal or family problems or practical difficulties (e.g. time, distance or transport).

6.6 Withdrawal rates

A total of 13 (11.0%) of participants withdrew from the study. Table 6.6 shows the pattern of withdrawals over the course of the study for the treatment group, control group and total sample. Participants were most likely to withdraw from the study in the first three months.

Table 6.6: Frequency and cumulative frequency of withdrawals from the study at each assessment point for the treatment group, control group and total sample.

		Pre-group	Post-group	6-Months	12-Months
Treatment group N=67	Frequency	3 (4.5%)	1 (1.5%)	3 (4.5%)	2 (3.0%)
	Cumulative Frequency	3 (4.5%)	4 (6.0%)	7 (10.4%)	9 (13.4%)
Control Group N=51	Frequency	1 (2.0%)	2 (3.9%)	0 (0.0%)	1 (2.0%)
	Cumulative Frequency	1 (2.0%)	3 (5.9%)	3 (5.9%)	4 (7.8%)
Total N=118	Frequency	4 (3.4%)	3 (2.5%)	3 (2.5%)	3 (2.5%)
	Cumulative Frequency	4 (3.4%)	7 (5.9%)	10 (8.5%)	13 (11.0%)

6.7 Comparison of participants who completed the study and those who provided no data or withdrew

It is important to establish whether there were any differences between the participants that completed the study and the participants who provided no data or withdrew during the study. Statistical analyses were carried out to test for differences between the 105 participants who remained in the study and the 31 participants who provided no data (n=18) or withdrew during the study (n=13). The proportion of people in the treatment or control groups, sex, age, duration of illness, and distance from the research centre were all compared (see Table 6.7).

In summary there were no significant differences between the group who provided no data or withdrew and those who remained in the study in the proportion of participants in the treatment and control groups, sex, duration of illness or distance from the research centre. The group who provided no data or withdrew from the study were significantly older than those who completed the study.

Table 6.7: Comparison of sex, age, duration of illness, distance from research centre, and experimental condition for participants who provided no data or withdrew and those that completed the study.

	No data/withdrawal	Completed study	Statistical test
N (% total)	31 (22.79%)	105 (77.21%)	
Sex	M = 10 (32.3%) F = 21 (67.7%)	M = 23 (21.9%) F = 82 (78.1%)	X ² = 1.396; df = 1; p = .237
Age	Mean 57.41 Range 39 – 69 s.d. 8.23	Mean 52.44 Range 22 – 69 s.d. 10.28	T = -2.41; df=134; P=.017*;
Duration of illness	Mean 12.53 Range 2 – 30 s.d. 9.27	Mean 14.08 Range 1 – 47 s.d. 10.17	T = .31; df = 115; p = .759
Distance from research centre	<15 = 19 61.3% >15 = 12 38.7% Median = 15–20 miles Mode = 5–10 miles	<15=67 63.8% >15=38 36.2% Median = 15-20 miles Mode = 5–10 miles	X ² =.065 df=1 p=.798
Exp Condition	Treatment = 18 (56.1%) Control = 13 (41.9%)	Treatment = 58 (55.2%) Control = 47 (44.8%)	X ² =.078; df=1 p=.781

Table 6.8: comparison of sex, age, duration of illness, distance from research centre and experimental condition for participants who withdrew and those that completed the study.

	Withdrew	Completed study	Statistical test
N (% total)	13 (11.02%)	105 (88.98%)	
Sex	M = 4 (30.8%) F = 9 (69.2%)	M = 23 (21.9%) F = 82 (78.1%)	X ² =0.515 df=1 p=0.473; Yates=0.135 df=1 p=0.713
Age	Mean 56.00 Range 39 – 69 s.d. 9.77 s.e.2.71	Mean 52.44 Range 22 – 69 s.d.10.28 s.e.1.01	t=-1.17 df=116 p=.246
Duration of illness	Mean 12.53 Range 2 – 30 s.d. 9.54 s.e. 2.65	Mean 14.08 Range 1 – 47 s.d.10.17 s.e. 1.00	t=.13 df=116 p=.895
Distance from research centre	<15 = 8 61.5% >15 = 5 38.5% Median = 15–20 miles Mode = 15–20 miles	<15=67 63.8% >15=38 36.2% Median = 15-20 miles Mode = 5–10 miles	X ² =0.026 df=1 p=0.872; Yates=0.00 df=1 p=1.00 00
Exp Condition	Treatment = 9 (69.2%) Control = 4 (30.8%)	Treatment = 58 (55.2%) Control = 47 (44.8%)	X ² =0.923 df=1 p=0.337; Yates=0.441 df=1 p=0.507

6.8 Comparison of participants who completed the study and those who withdrew during the study

Table 6.8 contains the results of statistical analyses comparing the 13 participants who withdrew from the study and the 105 participants who remained in the study. There were no differences between those who remained in the study and those who withdrew during the study with respect to the proportion of participants in the treatment and control groups, sex, age, duration of RA and distance from research centre.

A series of two-tailed independent samples T-tests were carried out to test whether there were any differences between people who withdrew and those who remained in the study on the main response variables. Only one significant difference was found, see Table 6.9. The group who withdrew from the study had a significantly lower mean self-efficacy for pain score, indicating that they were significantly less confident in their ability to manage their pain. As multiple comparisons were made, it is reasonable to use a stricter criterion for significance of $p < 0.01$. Using this criterion no results were significant. As some of the response variables did not meet assumptions for the use of parametric statistics, the analyses were also computed using a Mann-Whitney U test, but the findings were the same.

Table 6.9: Results of T-tests showing significant differences between participants who withdrew and those that completed the study

Variable	Completed N Mean (SD)	Withdrew N Mean (SD)	t	p	df
SE Pain	105 47.62 (19.50)	12 33.33 (11.67)	2.481	0.015*	115

The 13 participants who withdrew during the study gave the reasons listed in Table 6.10. Inspection of Tables 6.5 and 6.10 suggest that people in the treatment group are more likely to give time as their primary reason for providing no data or withdrawing from the study. It is possible that more people in the treatment group gave time as their reason because they were also offered the six treatment sessions. If this were the case we would expect most participants to drop out after the second assessment point, when they would have had the maximum additional demands on their time. However for those who gave time as a reason two participants dropped out before the first assessment point providing no data. For those who withdrew during the study, two withdrew after the first assessment point before they would have attended the treatment sessions. One withdrew after the third assessment, after the time period when they would have had to attend the treatment sessions. This suggests that being invited to attend the group sessions did not increase drop out from the study.

**PAGE
NUMBERING
AS ORIGINAL**

Table 6.18: Demographic characteristics of the sample (age, years of education, duration of illness and number of children and results of statistical analyses testing for differences between treatment and control groups.

Variable	Treatment			Control			Total			Statistics
	Mean	S.D.	Range	Mean	S.D.	Range	Mean	S.D.	Range	
Age	54.1	10.1	22-69	51.4	10.2	27-69	52.9	10.2	22-69	T=-1.414 (116) p=0.160
Years of Education	12.3	2.92	8-20	12.5	3.0	9-24	12.4	2.9	8-24	T=0.323 (107) p=0.747
Duration of Illness	13.52	10.13	1.5-47	13.12	10.05	1-36	13.35	10.05	1-47	T=-0.213 (115) p=0.831
No. of Children	2.26	1.50	0 - 7	1.65	1.13	0 - 4	2.0	1.38	0 - 7	T=-2.373 (112) p=0.019*

Table 6.19: Details of participants' marital status.

Marital Status	Treatment	Control	Total
Single	2 (3%)	5 (9.8%)	7 (5.9%)
Married	55 (82.1%)	38 (74.5%)	93 (78.8%)
Separated	1 (1.5%)	1 (2.0%)	2 (1.7%)
Divorced	7 (10.4%)	5 (9.8%)	12 (10.2%)
Widowed	2 (3.0%)	2 (3.9%)	4 (3.4%)

Table 6.20: Details of participants' employment status.

Employment	Treatment	Control	Total
Full-time	11 (16.4%)	16 (31.4%)	27 (22.9%)
Part-time	14 (20.9%)	8 (15.7%)	22 (18.6%)
Seeking Work	2 (3.0%)	2 (3.9%)	4 (3.4%)
Home Maker	10 (14.9%)	6 (11.8 %)	16 (13.6%)
Retired	16 (23.9%)	6 (11.8%)	22 (18.6%)
Disabled	14 (20.9%)	13 (25.5%)	27 (22.9%)

6.12 Research Question 1.1

RQ1.1 What are the baseline levels of disease activity, pain, physical function and emotional distress in this sample of people with RA?

The Treatment and Control groups and total sample means and standard deviations for the main response variables at baseline are shown in Appendix I.

Overall, the sample had moderate levels of disease activity which were consistent with other studies of chronic RA populations (Table 6.21). Abnormal levels of serum rheumatoid factor (sero-positive) were found in 66.4% of participants. This is similar to results found in other studies of chronic RA (Harris, 1993) CRP baseline results show that the majority of participants had mildly (74.5%) or moderately-severely (20.2%) active RA (Banks, Whicher, Thompson & Bird, 1998). The ESR results show that 86% of participants had active RA (above the normal range cut-offs for men 0-5mm/hour and female 0-7mm/hour).

Haemoglobin levels were below the normal range (males 13-18; females 11.5-16.5 10^{12} cells/litre) for 26.3% of participants. For WCC 8.8% were above the cut off (3.7-11 10^9 cells/litre). For Platelets 11.4% were above the cut off (140-450 10^9 cells/litre).

Table 6.21: Frequency and percentage of participants within and outside of normal ranges for blood tests.

CRP (Mg/l)	Normal < 10		Mild 10-39		Moderate – severe 40-200+	
N (%)	6	5.3%	85	74.5%	23	20.2%
	Male		Female		Total sample	
	Normal	Abnormal	Normal	Abnormal	Normal	Abnormal
ESR						
N (%)	2 8.3%	22 91.7%	14 15.6%	76 84.4%	16 14%	98 86%
HB						
N (%)	15 62.5%	9 37.5%	69 76.7 %	21 23.3%	84 73.7%	30 26.3%
	Normal Range			Above cut off		
WCC N (%)	104 91.2%			10 8.8%		
Platelets N (%)	101 88.6%			13 11.4%		

Participants reported moderately high levels of pain. The mean VAS pain intensity ratings (47-51) were higher than in other studies of chronic RA (27-33 Dixon *et al.*, 1988; 38 Parker *et al.*, 1988a). Participants also reported moderately high levels of early morning stiffness (mean EMS=113 minutes). This was similar to that reported by Dixon *et al.*, (1988) (mean EMS=112-242 minutes) but higher than that found by Parker *et al.*, (1988a) (EMS=72 minutes). Participants also reported a moderately high tender joint score (mean=15.1) which was a little lower than that reported by Dixon *et al.*, (1988) (means=19.8-25.9).

The mean HAQ score, 1.51, indicates a moderate to severe level of functional disability. Only 7.5% of participants had HAQ scores in the mild disability range 0-0.375. Wolfe (2001) found that 12.7% of their sample of people with early active RA, about to start a DMARD, had HAQ scores in this mild disability range. The majority of participants (81.4%) had HAQ scores in the moderate to severe disability range 1-3 (Table 6.22). The mean HAQ score of 1.51 in this sample is greater than that found in other studies of people with RA. For example, Wolfe (2001) reported the mean HAQ score for the sample of people with early active RA was 1.3. Uhlig *et al.*, (1998) report a mean HAQ score of 0.99 for a general RA sample. Wolfe, Hawley and Kathy (1991) found that people with a similar duration of RA (12-17 years) had a mean HAQ of 1.11.

Table 6.22: Frequency distribution for HAQ scores.

HAQ	<1	<2	<3
N (%)	22 18.6%	63 53.4%	33 28%

On the HADS the sample reported modest levels of anxiety and depression (HA mean=6.36 s.d.=4.09; HD mean=5.54 s.d.=3.24).

Table 6.23 and 6.24 show that 33% of the sample had scores indicating possible anxiety, 18% probable anxiety, 22% possible depression and 7% probable depression. The mean scores for anxiety are lower than, and those for depression similar to, those reported by Pincus *et al.*, (1996) in their UK RA sample (HA mean=8.1 s.d.=4.8; HD mean=6.2 s.d.=3.8). The prevalence rate for possible depression was similar (23%) but the rate for probable depression was higher (15%) in the Pincus *et al.*, (1996) study. However, the rates were much lower than those reported by Barlow *et al.*, (2002a) in a RA out-patient sample (possible anxiety=58% possible depression=35%).

Comparison with normative data for the HADS, for males and females separately, from a large UK sample (J. Henry, personal communication, October 28, 2002) shows that the mean anxiety scores are very similar (RA males mean=5.19 s.d.=4.45, females mean=6.7 s.d.=3.94; Norms males mean=5.55 s.d.=3.42, females mean=6.63 s.d.=3.95). The mean depression scores in the RA sample were higher than norms for both males and females (RA males mean=5.67 s.d.=3.85, females mean=5.51 s.d.=3.05; Norms males mean=3.51 s.d.=2.79 females mean=3.83 s.d.=3.29). The percentage of people in the normal range for depression (0-7) was lower than in the normative sample (RA males=82% females=77%; norms males=91.2% females=86.3%). In the RA sample there were more males in the moderate-severe range (11-21) (RA males=11%; norms males=2.2%) and more females in the mild range (8-10) than in the normative sample (RA females=18% norms=8.9%).

Overall, these results suggest that the sample has modestly elevated levels of depression compared to the normal population but lower than reported in other RA studies. Anxiety levels are similar to the normal population and lower than reported in other RA studies.

Table 6.23: HADS Anxiety Baseline frequency and percentage of participants below and above cut off for possible and definite anxiety by experimental group and gender.

HADS Anxiety	Treatment N (%)				Control N (%)				Total Sample N (%)			
	Male	Female	M&F		Male	Female	M&F		Male	Female	M&F	
Score 0-7	11 (69%)	32 (63%)	43 (63%)		9 (82%)	27 (67.5%)	36 (71%)		20 (74%)	59 (65%)	79 (67%)	
8-10	4 (25%)	8 (16%)	12 (18%)		0 (0%)	6 (15.0%)	6 (12%)		4 (15%)	14 (15%)	18 (15%)	
11-21	11 (6%)	11 (22%)	12 (18%)		2 (18%)	7 (17.5%)	9 (17%)		3 (11%)	18 (20%)	21 (18%)	
Total N (%)	16 (24%)	51 (76%)	67 (100%)		11 (22%)	40 (78%)	51 (100%)		27 (23%)	91 (77%)	118 (100%)	

Table 6.24: HADS Depression Baseline frequency and percentage of participants below and above cut offs for possible and definite depression by experimental group and gender.

HADS Depression	Treatment N (%)			Control N (%)			Total N (%)		
	Male	Female	M&F	Male	Female	M&F	Male	Female	M&F
Score 0-7	12 (75%)	40 (78%)	52 (78%)	10 (91%)	30 (75%)	40 (78%)	22 (82%)	70 (77%)	92 (78%)
8-10	2 (12.5%)	8 (16%)	10 (15%)	0 (0%)	8 (20%)	8 (16%)	2 (7%)	16 (18%)	18 (15%)
11-21	2 (12.5%)	3 (6%)	5 (7%)	1 (9%)	2 (5%)	3 (6%)	3 (11%)	5 (5%)	8 (7%)
Total N (%)	16 (24%)	51 (76%)	67 (100%)	11 (22%)	40 (78%)	51 (100%)	27 (23%)	91 (77.1%)	118 (100%)

Overall this sample of people with chronic RA have moderate levels of disease activity, high levels of pain, moderate-severe levels of physical disability and mildly elevated levels of depression, when compared to other RA samples and general population norms.

6.13 Research Question 1.2

RQ1.2 What are the illness representations of a sample of people with RA?

Descriptive statistics were computed to clarify the nature of participants' illness representations (Appendix I). Figure 6.1 presents the mean scores for each item of the Identity scale. On average participants perceive pain, loss of strength, stiff/sore joints, sleep problems and fatigue to be their most frequent symptoms. Figure 6.2 shows the mean and 95% confidence intervals for the IPQ Identity, Coherence, Time Line, Control/Cure, Consequences and Total Attributions scales. These data suggest that on average participants perceive their RA as having multiple, frequent and incoherent symptoms (variable, puzzling, and distressing), to be chronic, moderately controllable, have major life consequences and few causes. There are also large individual differences in people's scores on the IPQ scales.

Figure 6.3 shows the number of causes for their RA that participants identified at baseline. Most participants identified less than three causes. The number of participants attributing the cause of their RA to each type of cause is shown in Figure 6.4. The most common causes identified were fate/chance, genetic, stress, and germ/virus. Internal consistency was good for the Identity ($\alpha=0.733$) and Time Line ($\alpha=0.770$) scales. It was modest for The Coherence ($\alpha=0.582$), control/cure ($\alpha=0.419$) and consequences ($\alpha=0.495$) scales. It was poor for the Total Attributions ($\alpha=0.304$) scale. The low internal consistency of the Total Attributions scale is not surprising given that it is made up of disparate items. Weinman *et al.*, (1996) suggested that it is not appropriate to combine cause items into a scale, as they do not measure a unitary construct.

Figure 6.1.: Graph showing control, treatment & total sample mean & 95% confidence intervals for symptoms reported on IPQ Identity at time 1

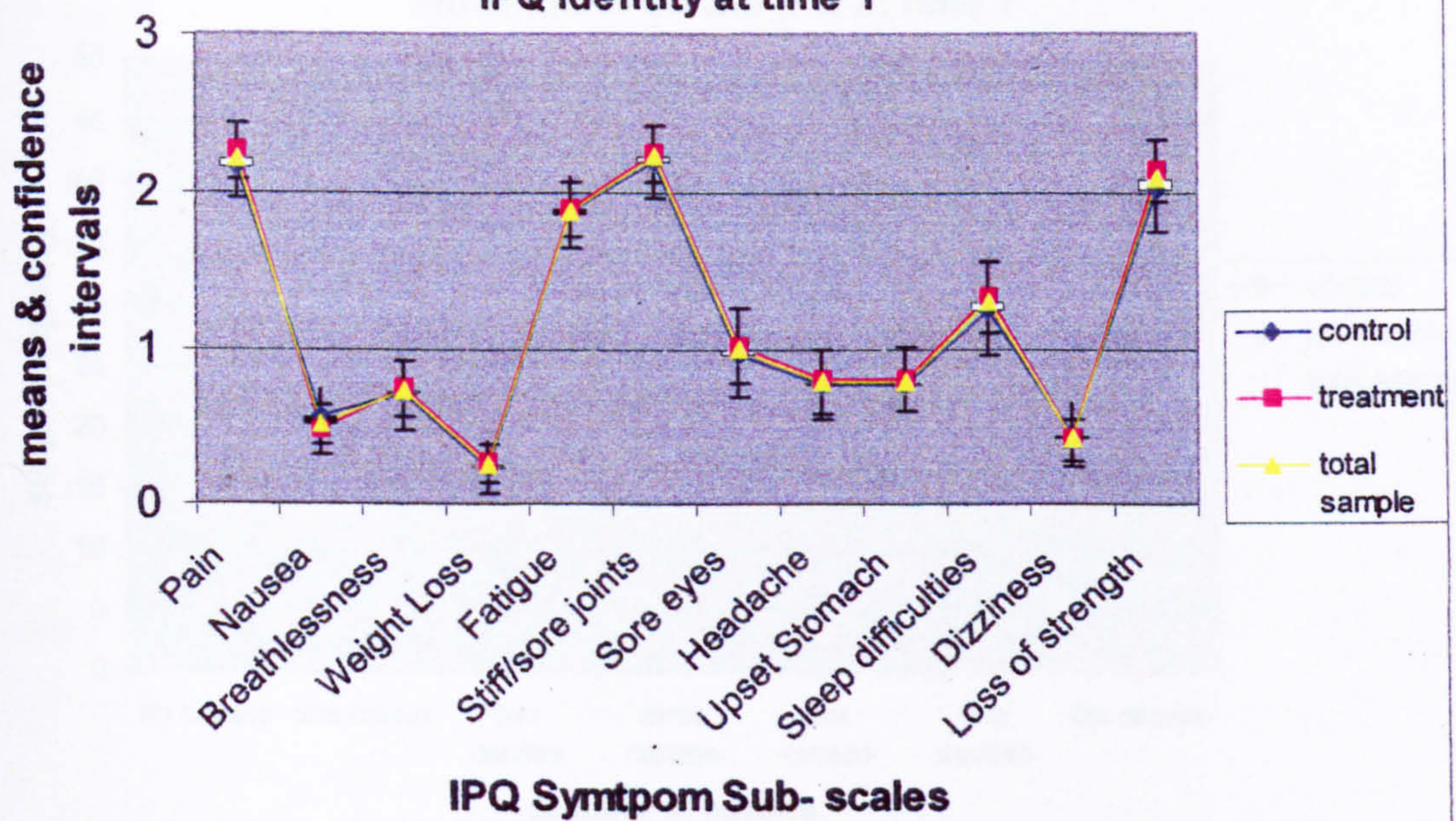


Figure 6.2.: Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ scales at time 1

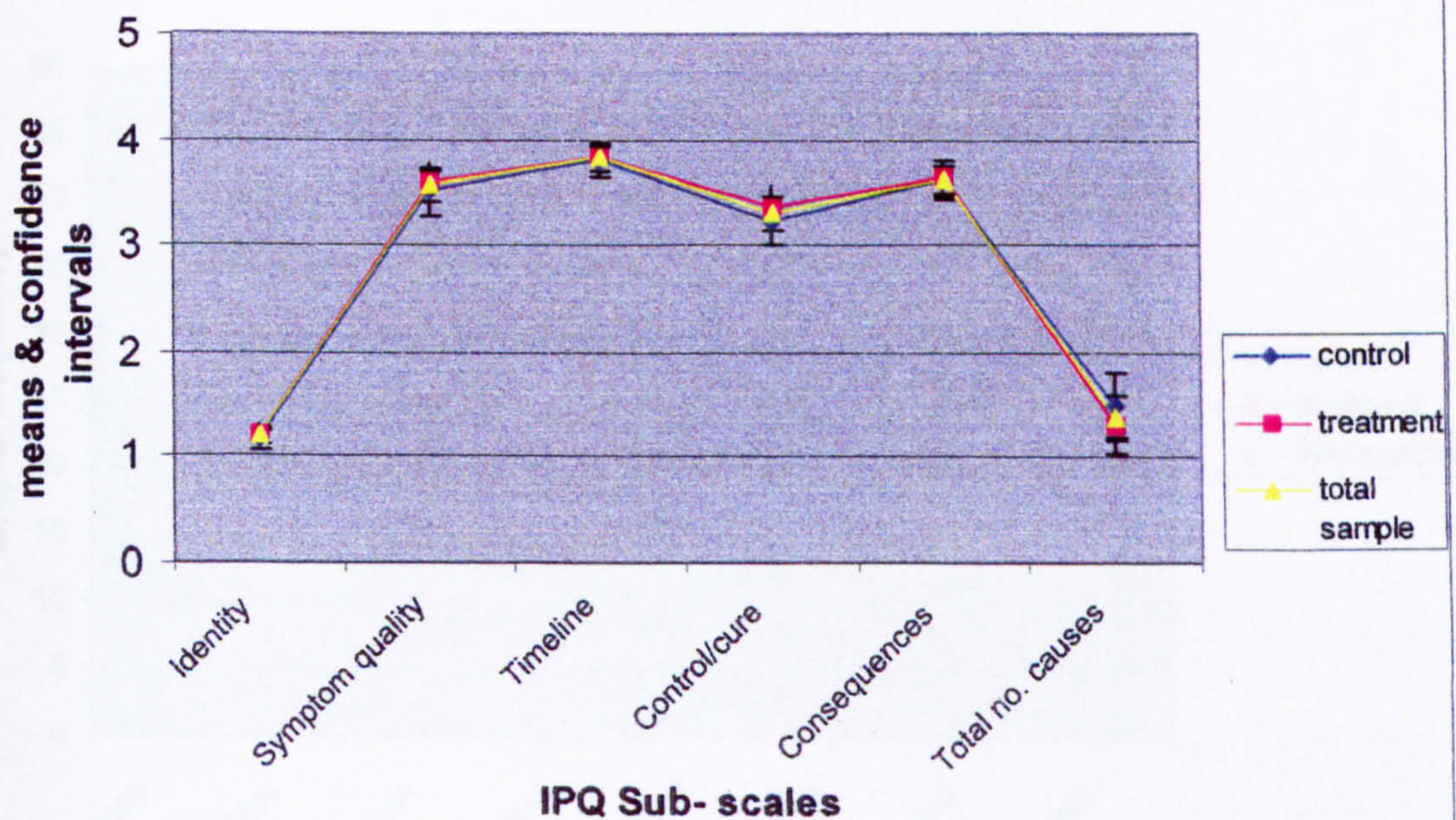


Figure 6.3.: Graph showing frequency of control, treatment & total sample participants reporting different numbers of causal attributions on the IPQ at time 1

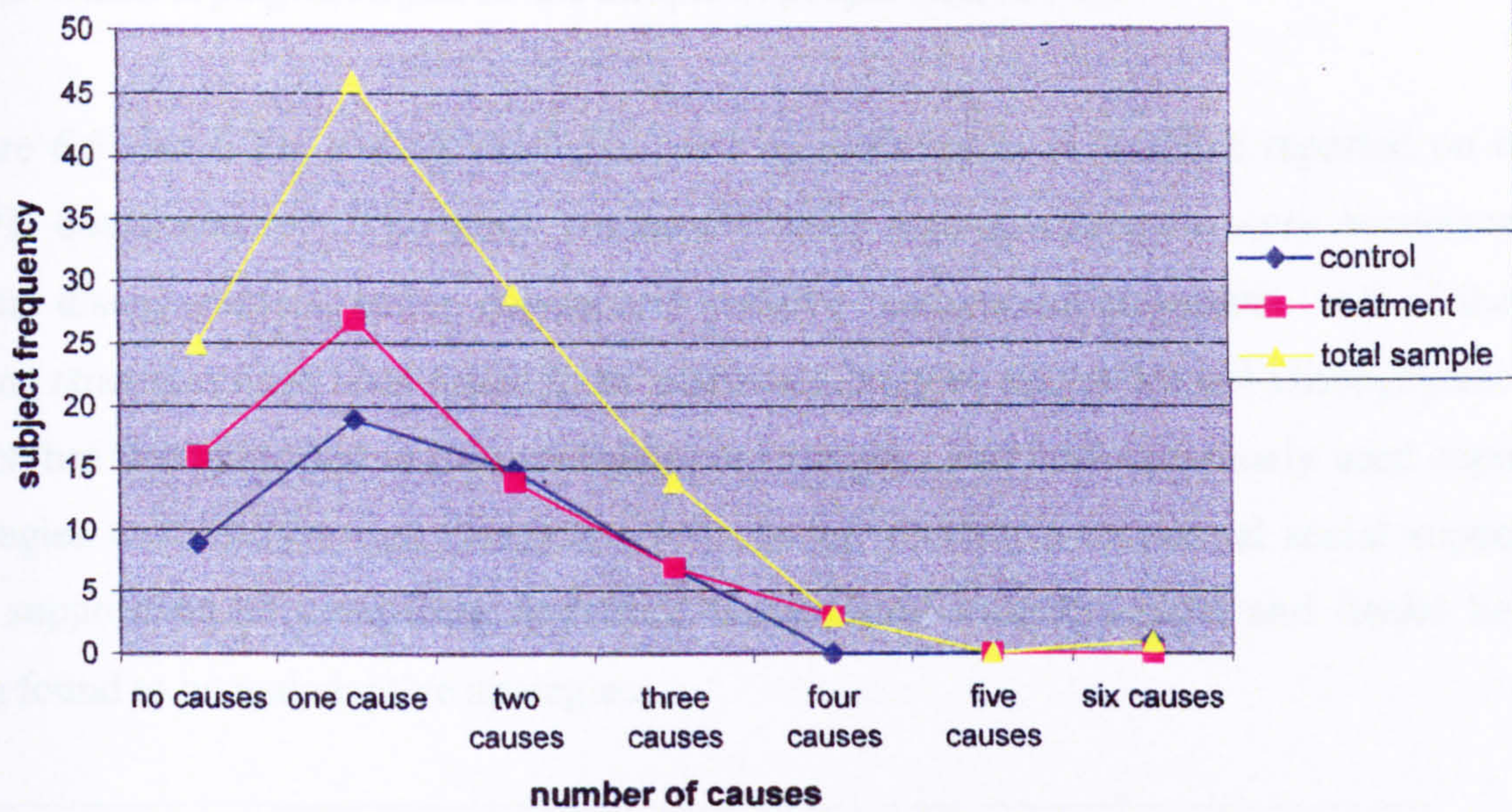
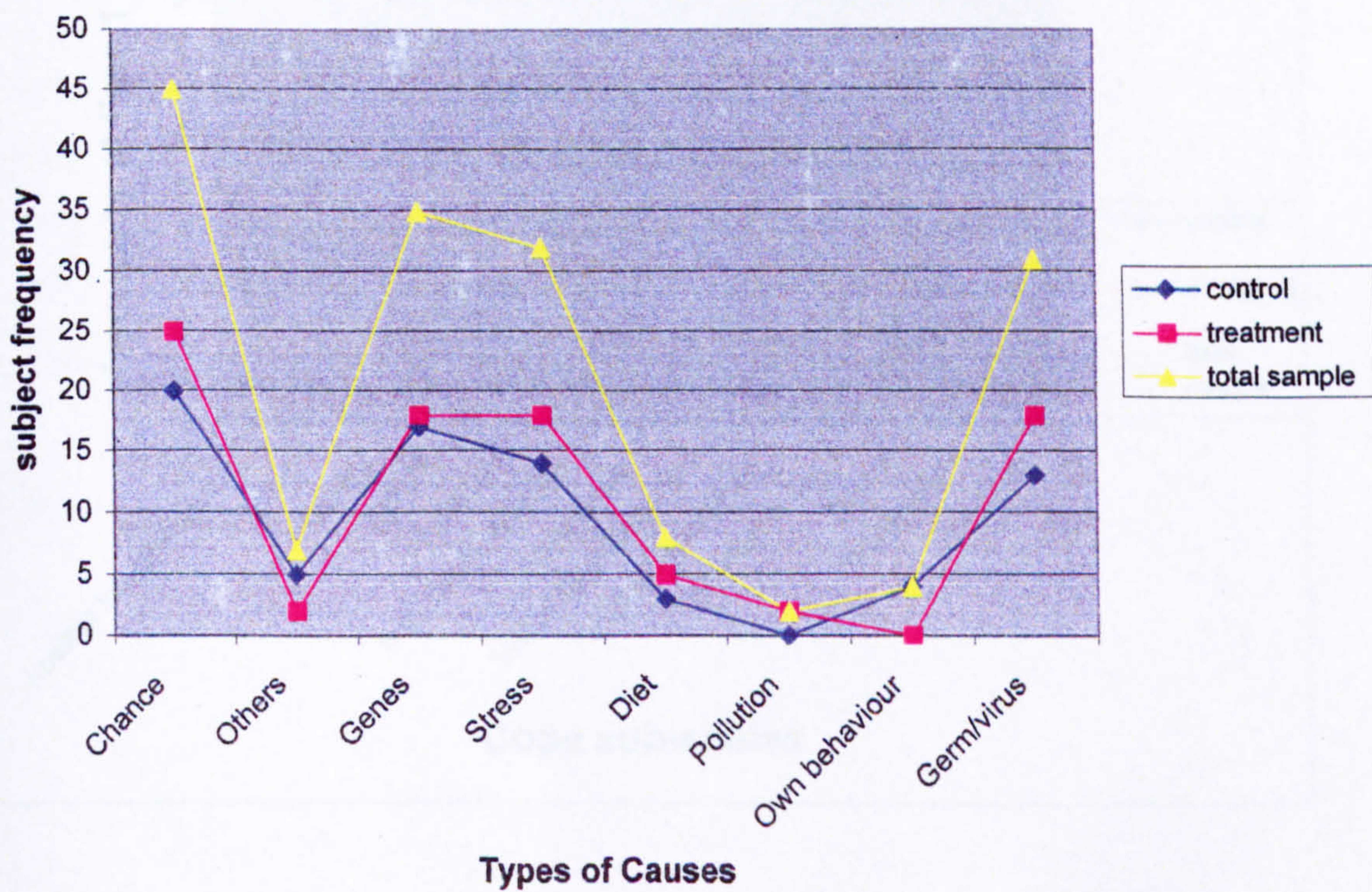


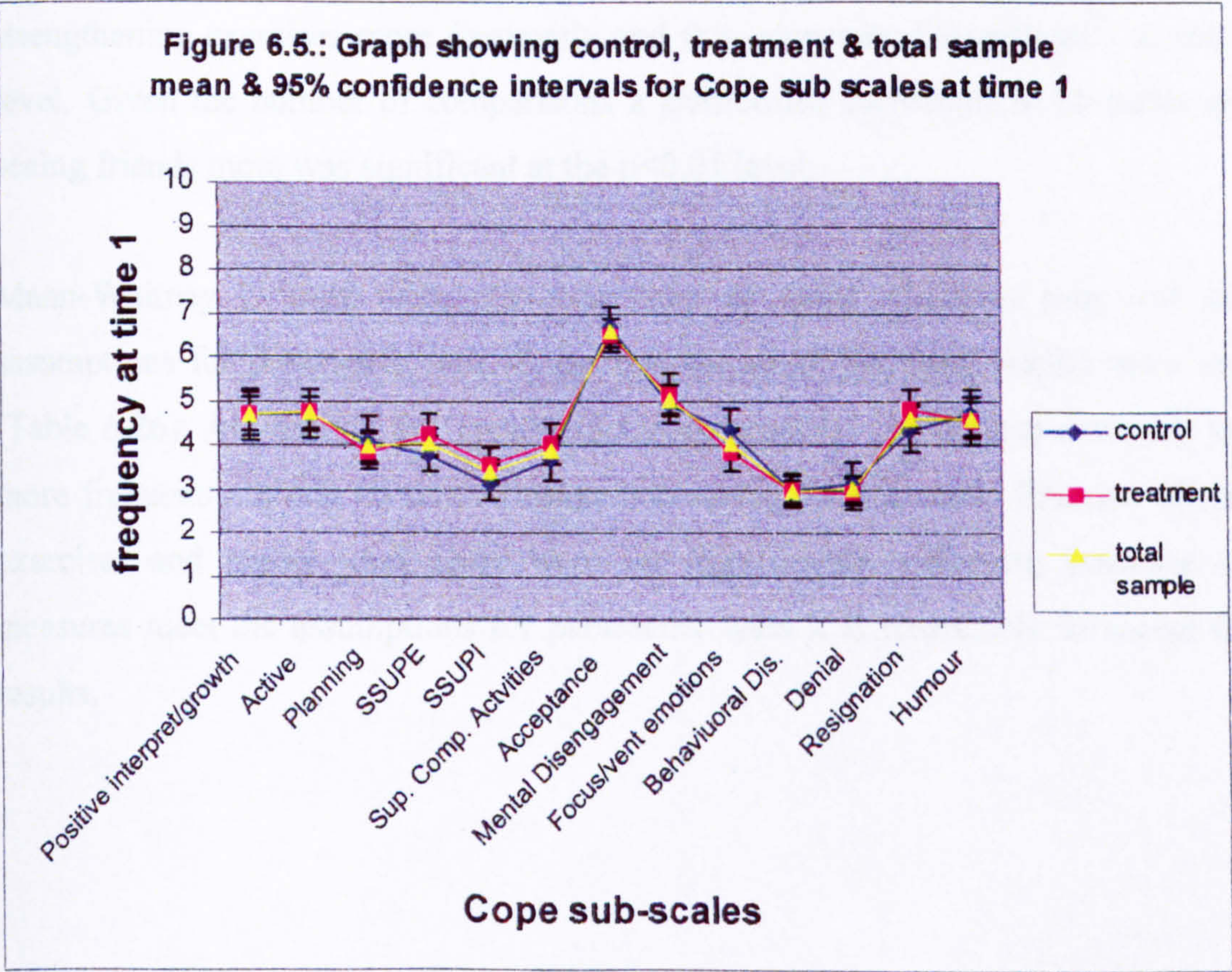
Figure 6.4.: Graph showing frequency of control, treatment & total sample participants reporting different types of causes at time 1



6.14 Research Question 1.3

RQ1.3 Which coping strategies do the sample of people with RA use?

Figure 6.5 shows the coping strategies used by participants at baseline reported on the COPE questionnaire. The most commonly used coping strategies were acceptance, mental disengagement, active coping and positive reinterpretation/growth. All of these coping strategies have been found to be adaptive strategies except Mental Disengagement which has been reported to be a maladaptive strategy. The least commonly used coping strategies were behavioural disengagement, denial, seeking instrumental social support, and suppression of competing activities. Behavioural disengagement and denial have been found to be maladaptive strategies.



6.15 Comparison of treatment and control groups at baseline on main response variables

Two-tailed independent samples t-tests and Mann-Whitney U tests were performed to compare treatment and control groups on the main response variables at baseline (Appendix I). There were a number of differences between the treatment and control groups at the $p < 0.05$ level which are shown in Table 6.25. The t-tests show that at baseline the treatment group were using stretching exercises, going on more outings (Outings), seeing friends (Social) and performing everyday activities more frequently than the control group. The Outings and Social measures are two of the four subscales of the Everyday Activities scale. The treatment group had a lower tender joint score (Ritchie Articular Index) than the control group. The Treatment group also used strengthening exercises more frequently and this approached significance at the $p = 0.05$ level. Given the number of comparisons a Bonferroni correction is advisable and only seeing friends more was significant at the $p < 0.01$ level.

Mann-Whitney U tests were also computed as some variables may not meet the assumptions for parametric tests. Some but not all of the t-test results were replicated (Table 6.26). At baseline the Treatment Group reported performing everyday activities more frequently, going on more outings and seeing friends more. Practice of stretching exercises and tender joint score were not significantly different, however as these measures meet the assumptions for parametric tests it is reasonable to accept the t-test results.

Table 6.25: Significant T-test results comparing control and treatment groups on baseline means of main response variables.

Variable	N Mean (SD) Treatment	N Mean (SD) Control	Leven's F p	t	p	df
Stretch	67 3.701 (5.524)	50 2.11 (3.08)	4.646 0.033*	-1.982	0.05*	107.3
Strength	67 2.28 (4.07)	50 1.11 (2.30)	10.21 0.002**	-1.977	.051	107.9
Everyday Activities	67 2.49 (.89)	50 2.073 (0.85)	Ns	-2.57	0.011*	115
Outings	67 2.55 (1.13)	50 2.11 (1.05)	Ns	-2.146	0.034	115
Social	67 2.14 (1.10)	50 1.63 (0.95)	Ns	-2.629	0.01**	115
Ritchie Articular Index	63 13.0 (10.07)	51 17.77 (13.21)	6.331 0.013*	2.124	0.036*	91.7

Table 6.26: Significant Mann-Whitney U results comparing control and treatment groups on baseline means of main response variables

Variable	Mean (SD) Treatment	Mean (SD) Control	U	W	z	P
Everyday Activities	2.492 (0.888)	2.073 (0.847)	1232	2507	-2.441	0.015*
Outing	2.551 (1.133)	2.11 (1.054)	1320.5	2595.5	-1.958	0.05*
Social	2.138 (1.101)	1.628 (0.945)	1224.5	2499.5	-2.488	0.013*

6.16 Sample characteristics and representativeness: summary of findings

Table 6.27 summarizes the results of the comparison of participants who completed the study and those who declined to participate, provided no data, withdrew or did not engage in treatment. In summary study participants were representative of people with RA attending the out-patient clinic who were eligible to participate. However study participants were significantly more likely to live nearer the research centre than those who declined to participate. There were few differences between participants who completed the study and those who provided no data or withdrew. The 18 participants who provided no data were significantly older and had had RA longer than the 118 participants who provided data. The 31 participants who provided no data or withdrew from the study were significantly older than the 105 participants that completed the study. The 13 participants who withdrew from the study had significantly lower self-efficacy for pain indicating that they were less confident in their ability to manage their pain than the 105 participants who completed the study. A significantly higher proportion of participants withdrew from the study in the group that did not complete treatment than in the group that completed treatment. The group who completed treatment had a significantly higher mean score for frequency of performance of household tasks at the start of the study.

Table 6.27. Summary of results of statistical analyses comparing study participants and those who declined to participate, provided no data, withdrew or did not complete treatment and treatment and control groups.

Participants	Non-participants	Total	Statistically Significant differences
136	264	400	Distance
Provided Data	Provided No Data		
118	18	136	Age Duration RA
Completed Study	Provided no data or Withdrew		
105	31	136	Age
Completed study	Withdrew		
105	13	118	SE Pain
Completed treatment	Not Completed Treatment		
47	20	67	Withdrawal rate, Household Tasks (Chores)
Treatment	Control		
67	51	118	Number of children, stretching exercises, social, outings, everyday activities, Ritchie

With respect to the demographic characteristics of the sample of 118 participants who provided data 77.1% were female (sex ratio 3:1) and the mean age was 52.9. The majority of participants were married, 78.8% and they had a mean 2.0 children. They had

a mean 12.4 years of education. A minority, 41.5% were in paid employment. All participants described their ethnic origin as "white UK" or "white european". The mean duration of RA was 13.4 years.

RQ1.1 What are the baseline levels of disease activity, pain, physical function and emotional distress in this sample of people with RA?

Overall this sample of people with chronic RA have moderate levels of disease activity, high levels of pain, and moderate-severe levels of physical disability when compared to other RA studies. Levels of depression were elevated compared to general population norms but were lower than reported for other RA studies. Anxiety levels are similar to the normal population and lower than reported in other RA studies.

RQ1.2 What are the illness representations of a sample of people with RA?

On the IPQ Identity scale participants perceive their RA as having a strong illness identity (multiple and frequent symptoms). The most frequent symptoms attributed to RA were pain, loss of strength, stiff/sore joints, sleep problems and fatigue.

On the other IPQ scales, on average, participants perceived RA as having incoherent symptoms (variable, puzzling, and distressing), to be chronic, moderately controllable, have major life consequences and few causes. Most participants identified less than three causes for their RA. The most common causes identified were fate/chance, genetic, stress, and germ/virus. There were also large individual differences in people's scores on the IPQ scales.

RQ1.3 Which coping strategies do the sample of people with RA use?

On the COPE the most commonly used coping strategies were acceptance, mental disengagement active coping, and positive reinterpretation/growth. All of these coping strategies have been found to be adaptive strategies except mental disengagement which has been reported to be a maladaptive strategy. The least commonly used coping

strategies were behavioural disengagement, denial, seeking instrumental social support, and suppression of competing activities. Behavioural disengagement and denial have been found to be maladaptive strategies.

Comparison of the treatment and control group showed that the treatment group had significantly more children than the control group. At baseline the treatment group were using stretching exercises, going on more outings (Outings), seeing friends (Social) and performing everyday activities more frequently than the control group. The Outings and Social measures are two of the four subscales of the Everyday Activities scale. The treatment group had a lower tender joint score (Ritchie Articular Index) than the control group.

Chapter 7: The efficacy of a self-management intervention for people with chronic RA

7.1 Statistical methods

A series of two way repeated measures Analyses of Variance were computed to test Research Questions 1.4 and 1.5 and hypotheses 1.1 and 1.2. The entire sample of 118 participants who had provided data were included in the analysis with missing data being handled using the methods described in Chapter 6. As this was an Intention-to-Treat design all participants in the treatment group were included even if they did not receive the treatment.

The measures were assumed to be interval quality data. The other assumptions for repeated measures ANOVA were checked by first computing Kolmogorov-Smirnov tests for the treatment and control groups at each time point on the main response variables to establish whether the distribution of scores for the treatment and control groups were significantly different from normal. A very small number of variables were found to have distributions significantly different from normal at one or more time points. ANOVAs assume normality of measurements but they are robust to departures from normality. Levene's test for homogeneity of variance were computed to ensure that the variances of the treatment and control group were not significantly different at each time point. There were a small number of variables where the Levene's test was significant at one or more time points. Most notably the HAQ time points 4 and 5 and the Ritchie Index at all time points. As the ANOVA is robust it was decided to continue. However, as there was a consistent difference on the Ritchie Index, the results of this ANOVA need to be interpreted with caution. The number of ANOVA's conducted on the same data set increases the risk of a type one error.

The Greenhouse-Geisser statistic was used for the Within Participants Effects as it is the most conservative statistic. To aid interpretation of the ANOVAs, planned contrasts were computed for time 1 vs 3, 1 vs 4, 1 vs 5, 1 vs 2, 2 vs 3, 3 vs 4, and 4 vs 5. The ANOVA is used to identify the overall pattern of results over the course of the entire study. The planned contrasts are used to identify whether changes occur between specific points in time. The planned contrasts for time 1 vs 3, 1 vs 4, 1 vs 5 establish whether change occurs immediately following treatment and if it is maintained. The planned contrasts for 1 vs 2, 2 vs 3, 3 vs 4, and 4 vs 5 establish the patterns of change occurring during the study that may be masked by the overall ANOVA results. These patterns may have important clinical implications and so significant planned contrasts are reported even if the overall ANOVA has no significant main or interaction effects. As a significant between groups main effect was found, for the Ritchie Index, Analyses of Covariance, using the Ritchie Index score at time 1 as the covariate, were also computed to test hypotheses 1.1 and 1.2 controlling for any difference between the groups.

There was evidence of significant change in disease activity (CRP) during the study. To control for the potential effect of this on the outcome variables, ANOVAs were carried out on the residualized dependent variables (i.e. the variance due to CRP was partialled out of the dependent variable at each time point (Llabre et al., 1991). A further ANCOVA was also conducted which controlled for both the Ritchie Index at the start of the study and the change in CRP over time. The results of ANCOVAs are only reported if they are significantly different from the results of the ANOVAs. The results of the ANOVAs and ANCOVAs are reported in Appendix J, tables J.1. – J.6. Graphs were plotted for the intervention and control groups and total sample means and 95% confidence intervals on the main response variables over time to aid interpretation of the results.

7.2 Changes in health outcomes following intervention

The first analyses addressed Research Question 1.4 And Hypothesis 1.1.

RQ1.4 How do health outcomes in a sample of people with RA change over one year?

Hypothesis 1.1 The intervention will produce a significant improvement in the following health outcomes immediately after intervention, at 3-months and 9-month follow up.

7.2(a) Pain.

On the GRSP, BVAS, NRIP, pain DISTRESS and AIMS Symptoms scales no significant main or interaction effects were found on the ANOVA. Therefore the hypothesis is rejected.

The planned contrasts reveal significant changes in pain immediately following the intervention on several measures. However these are not maintained at follow up. For GRSP Figure 7.2 shows that the treatment group's pain scores decreased between time 1 and 3. This is confirmed by a significant within participant main effect for the planned contrast time 1 vs 3 ($F = 4.94$, $df=1$, $p=0.028^*$) and a significant interaction effect for the planned contrast 1 vs 3 ($F = 4.573$, $df=1$, $p=0.035^*$). The Treatment group's pain scores return to slightly better than baseline levels at time 4. This is confirmed by a significant interaction effect for the planned contrast time 3 vs 4 ($F=4.41$, $df=1$, $p=0.04^*$). In summary the treatment group's pain improves between time 1 and 3 and then returns to slightly better than baseline levels of pain by time 4. This is maintained at time 5. The control group's pain scores show little change over time.

On AIMS Symptoms Figure 7.5 shows that the treatment group's pain scores improved between time 1 and 3. This is confirmed by a significant within participant main effect for the planned contrasts time 1 vs 3 ($F = 7.471$, $df=1$, $p=0.007^{**}$) and a trend towards a significant interaction effect ($F = 3.895$, $df=1$, $p=0.051$). The Treatment group pain scores return to slightly better than baseline levels of pain at time 4 which is confirmed by the planned contrast for time 3 vs 4 for which there was a significant within participants main effect ($F=3.94$, $df=1$, $p=0.05^*$) and a significant interaction effect ($F = 3.94$, $df=1$, $p=0.05^*$). In summary the treatment group's pain scores (AIMS Symptoms) improve between time 1 and 3. They return to slightly better than baseline levels of pain by time 4 and this is maintained at time 5. The control group shows little change.

On the Pain Distress VAS there was a significant within participants main effect for the planned contrast 1 vs 4 ($F = 4.135$, $df=1$, $p=0.044^*$). Figure 7.4 shows that for the whole sample there was an improvement in pain distress scores between time 1 and 4 with a slight increase in pain distress at time 5. The treatment group improved between time 1 and 3. Their pain distress scores increase at time 4 and 5 although they are still slightly better than baseline. The control group's pain distress scores improve between time 3 and 4 and this is maintained at time 5.

For the BVAS there was a significant interaction effect for the planned contrast time 3 vs 4 ($F = 4.57$, $df=1$, $p=0.03^*$). Figure 7.3 shows that on the BVAS the treatment group's pain scores improved following the intervention. During the follow up period the treatment group's pain scores increased to slightly above the baseline level. The control group's pain scores decreased between time 2 and 4. Their scores at time 5 were lower than their baseline levels. In summary the interaction effect is due to an increase in pain for the treatment group and a decrease in pain for the control group between time 3 and 4.

Figure 7.1: Graph showing control, treatment & total sample mean & 95% confidence intervals for NRIP scores between time 1 & 5

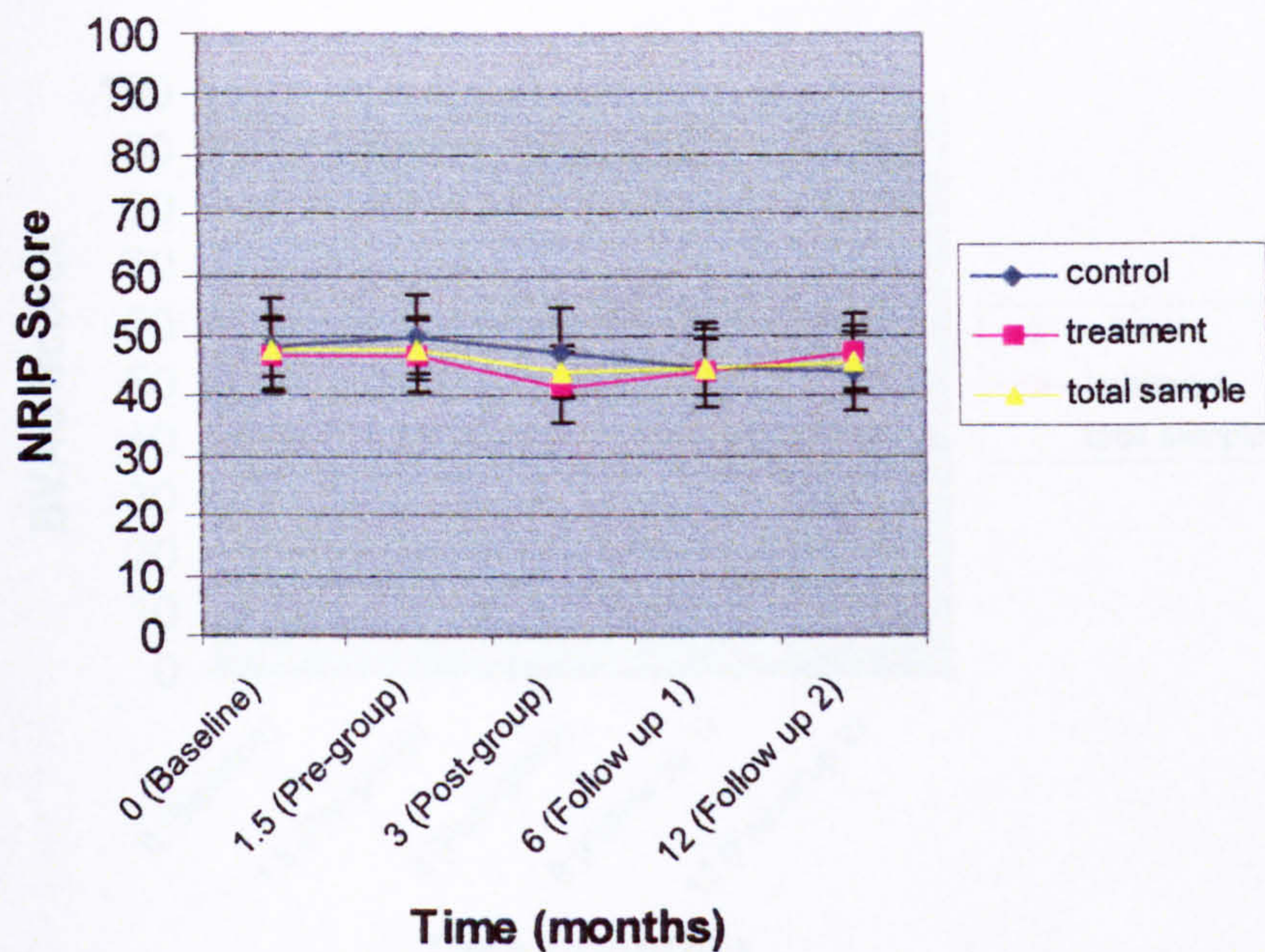


Figure 7.2: Graph showing control, treatment & total sample mean & 95% confidence intervals for GRSP scores between time 1 & 5

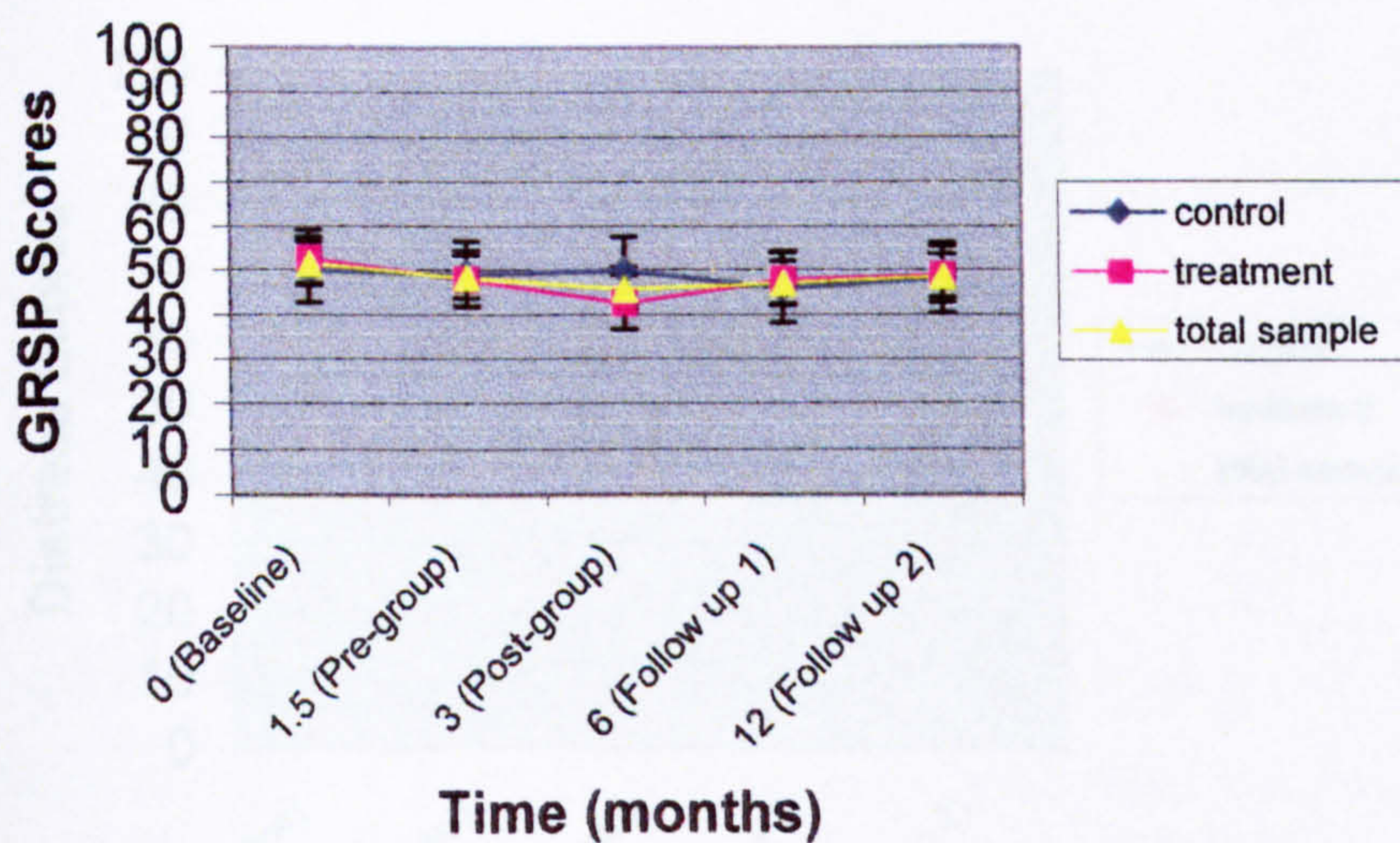


Figure 7.3: Graph showing control, treatment & total sample mean & 95% confidence intervals for BVAS scores between time 1 & 5

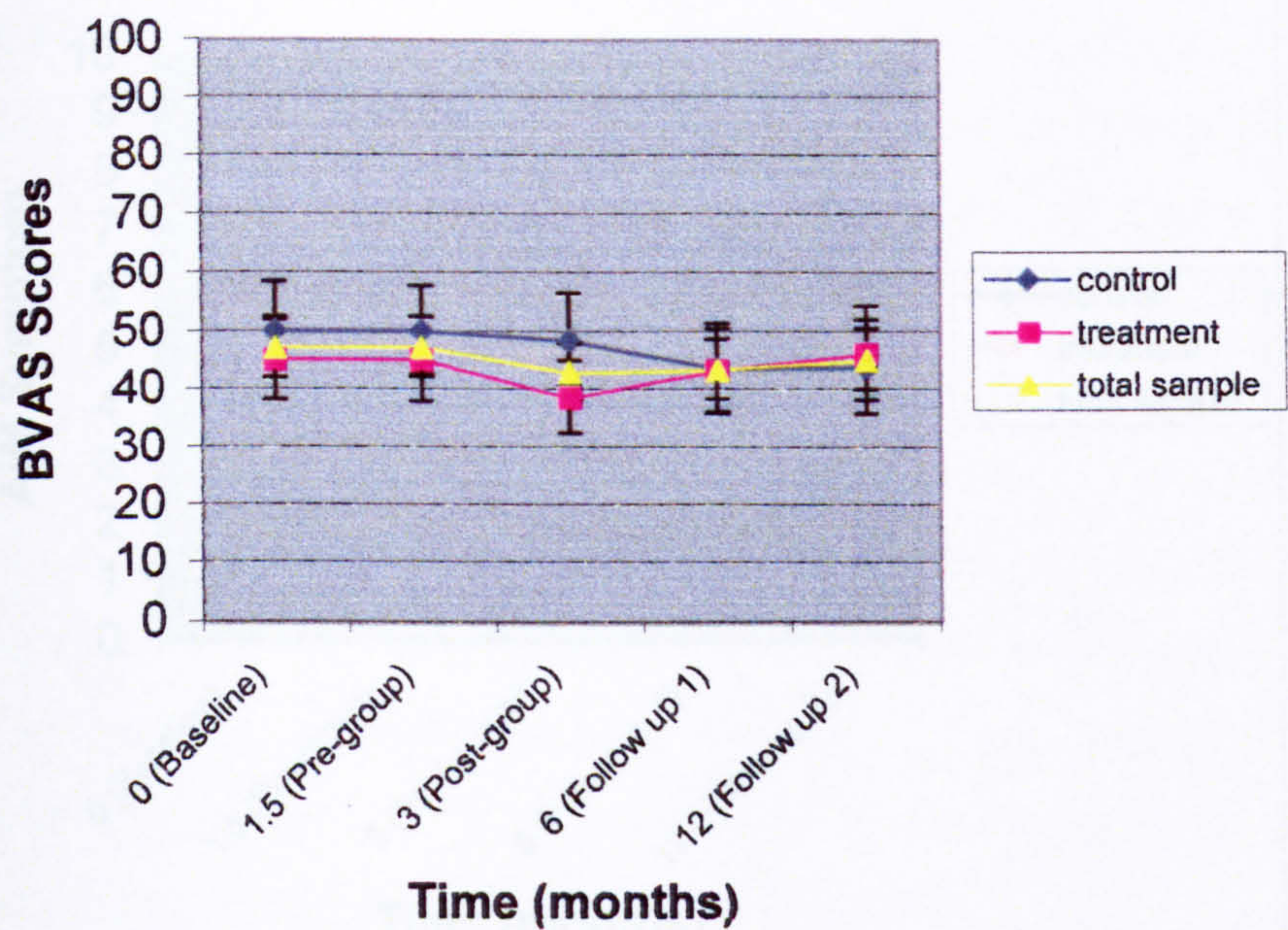


Figure 7.4: Graph showing control, treatment & total sample mean & 95% confidence intervals for pain distress scores between time 1 & 5

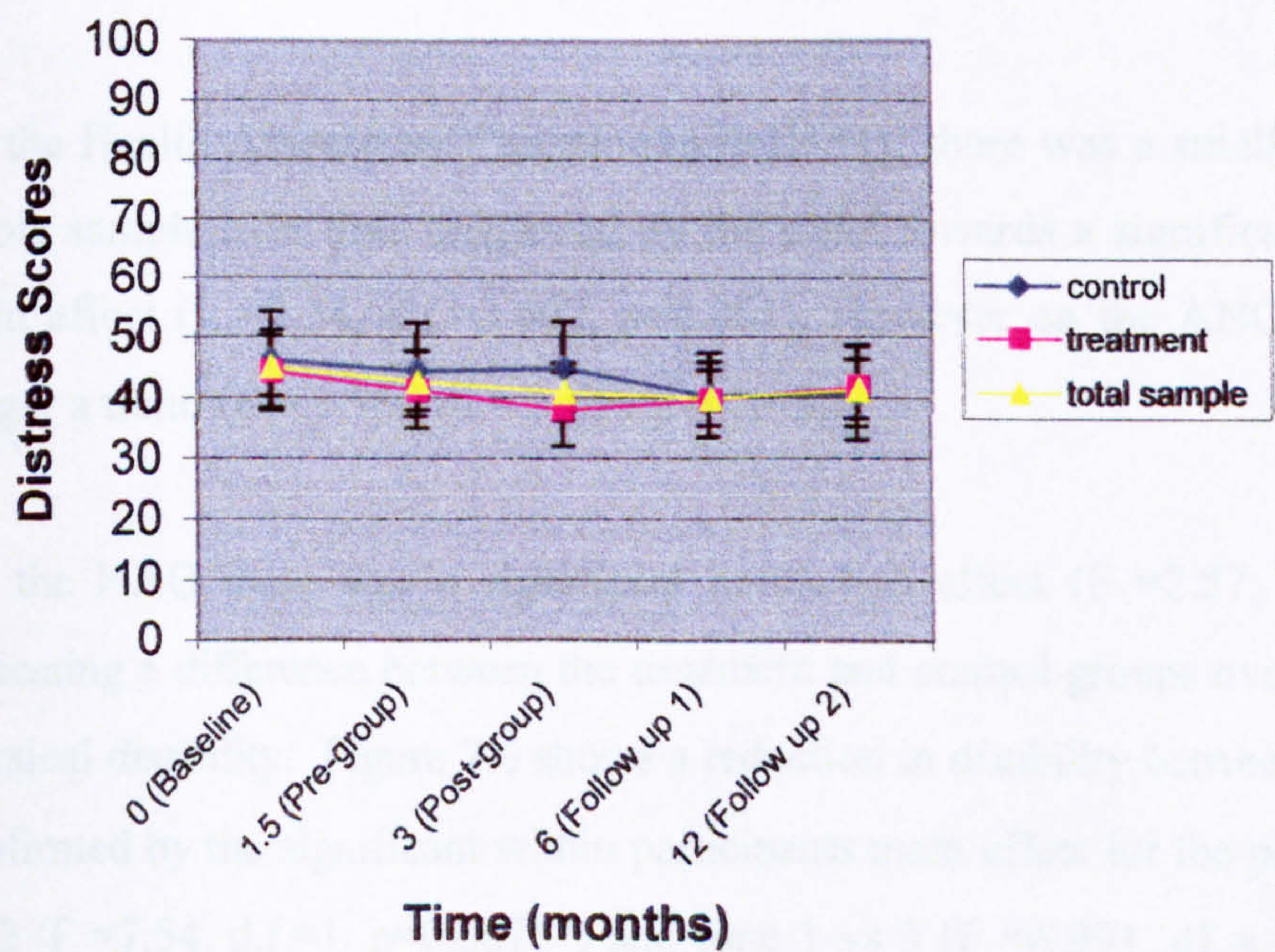
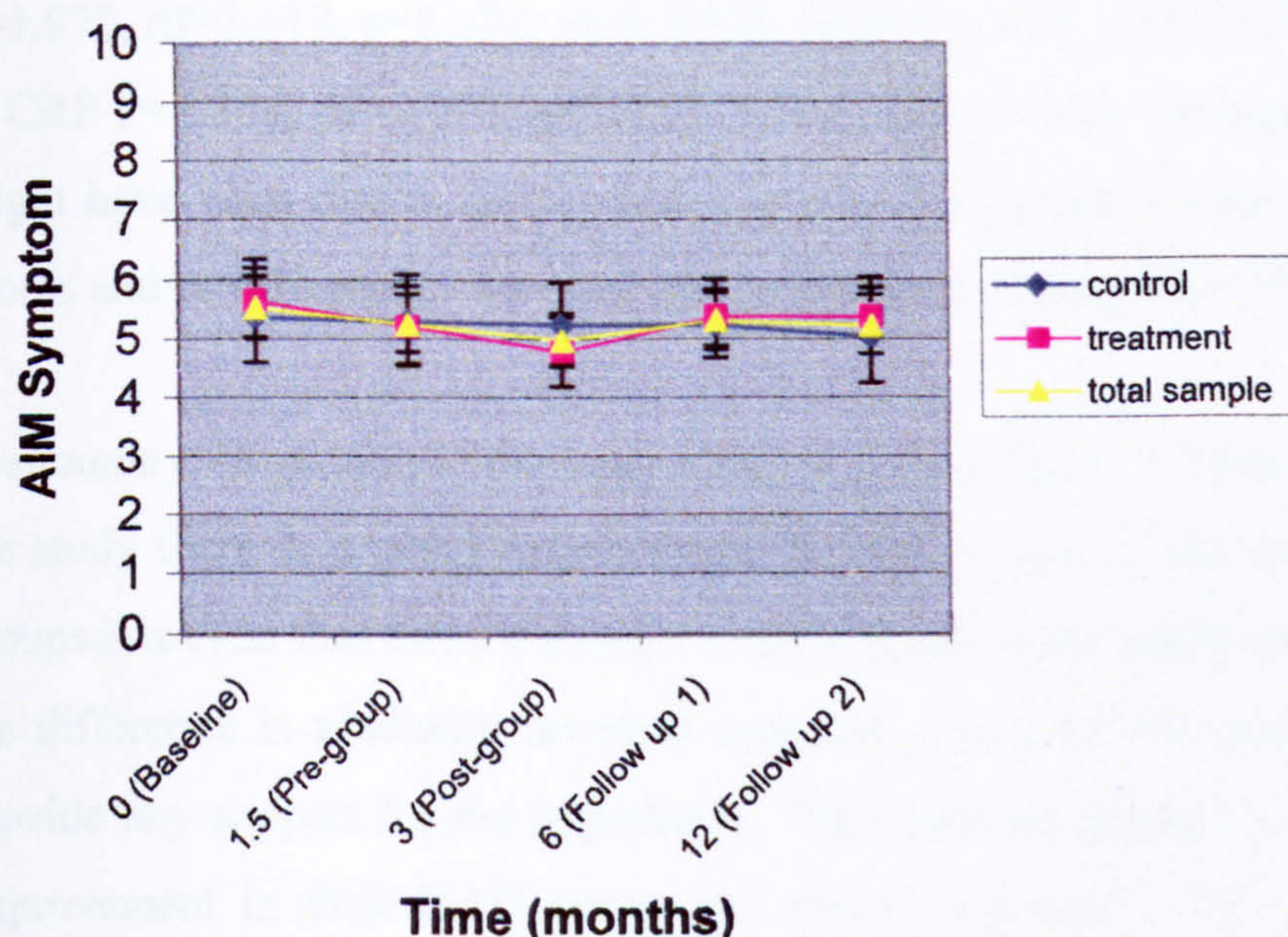


Figure 7.5: Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Symptom scores between time 1 & 5



7.2(b) Physical disability

On the Health Assessment Questionnaire (HAQ) there was a small improvement for the whole sample over time suggested by the trend towards a significant within participants main effect ($F = 2.34$, $d.f. = 3.607$, $p = 0.061$). However on the ANCOVA RI this was no longer a trend ($F = 1.760$ $df = 3.619$ $p = .143$).

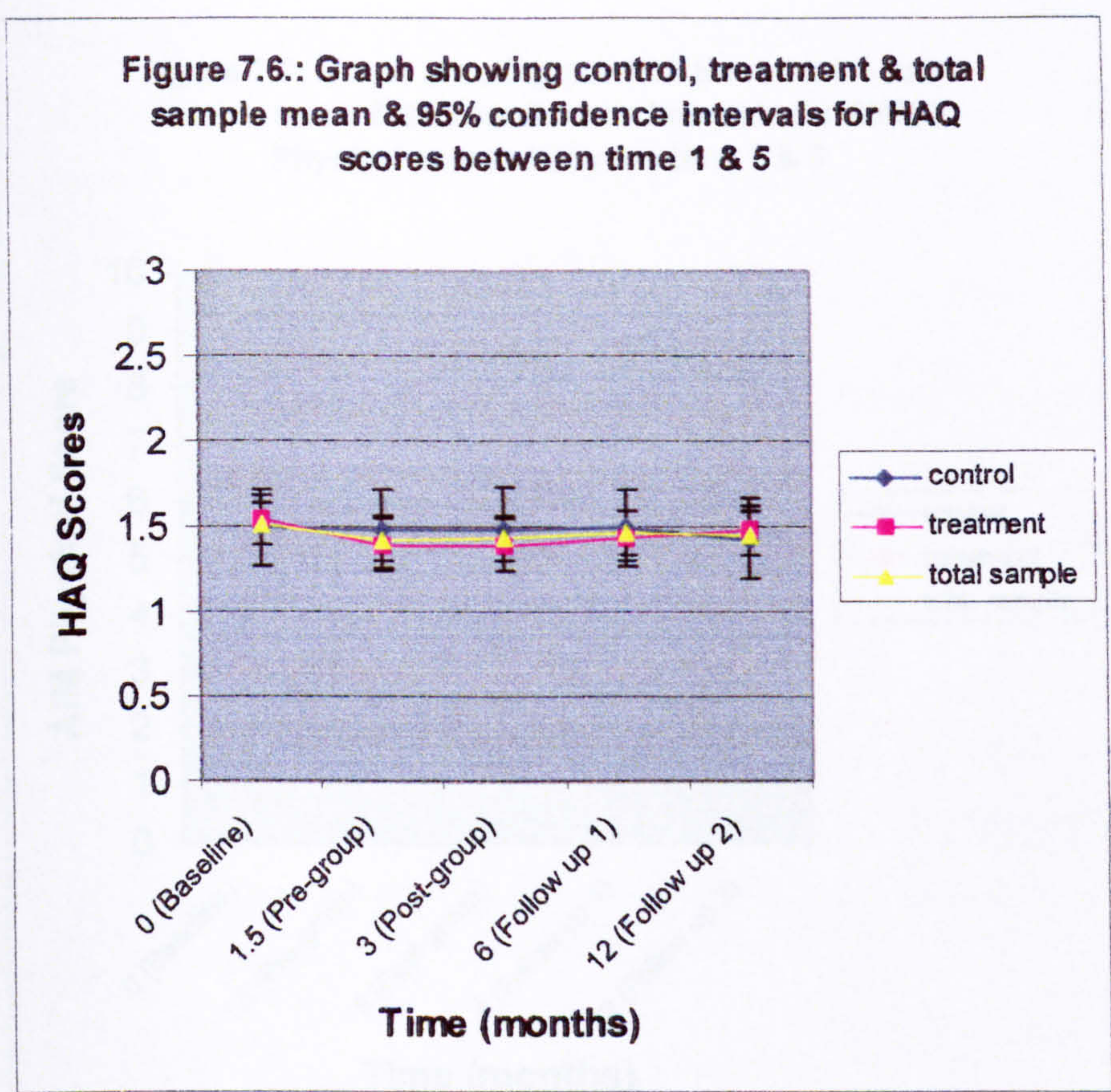
On the HAQ there was a significant interaction effect ($F = 2.57$, $d.f. = 3.607$, $p = 0.044^*$) indicating a difference between the treatment and control groups over time in their level of physical disability. Figure 7.6 shows a reduction in disability between time 1 and 2. This is confirmed by the significant within participants main effect for the planned contrasts time 1 vs 2 ($F = 7.54$, $d.f. = 1$, $p = 0.007^{**}$) and time 1 vs 3 ($F = 6.991$, $df = 1$, $p = .009^{**}$). Figure 7.6 shows that most of the change occurred in the treatment group. This is confirmed by

planned contrasts which show significant interaction effects between time 1 and 2 ($F = 5.46$, $df=1$, $p = 0.021^*$) and time 1 and 3 ($F = 5.057$, $df = 1$, $p = .026^*$).

However none of the ANCOVAs showed a significant interaction effect (ANCOVA RI $F=1.870$, $df=3.619$, $p=0.122$; ANCOVA CRP $F=1.698$, $df=3.697$ $p=0.155$, ANCOVA RI & CRP $F=1.359$, $df=3.703$, $p=0.250$). This suggests that the significant interaction effect might have been due to differences between the groups at time 1 in their Ritchie Index scores and/or differences between the groups in the change in CRP during the study.

In summary, inspection of the mean HAQ scores in Figure 7.6 shows that over the course of the study there is a small improvement in both groups HAQ scores. Comparing the two groups it is clear that there is a larger improvement in the treatment group from time 1-2 but the difference is gradually reversed over the course of the study. Overall this does not provide any support for the hypothesis. The treatment group shows a significantly greater improvement in their HAQ scores but this occurs before the treatment. The difference between the treatment and control groups disappears by 12 months and when differences in initial RI and CRP change are controlled for.

There were no significant results on the ANOVA or planned contrasts for the AIMS Physical scale. On Figure 7.7 the treatment group shows a slight improvement between time 1 and 3 and then returns to baseline and the control group shows little change.



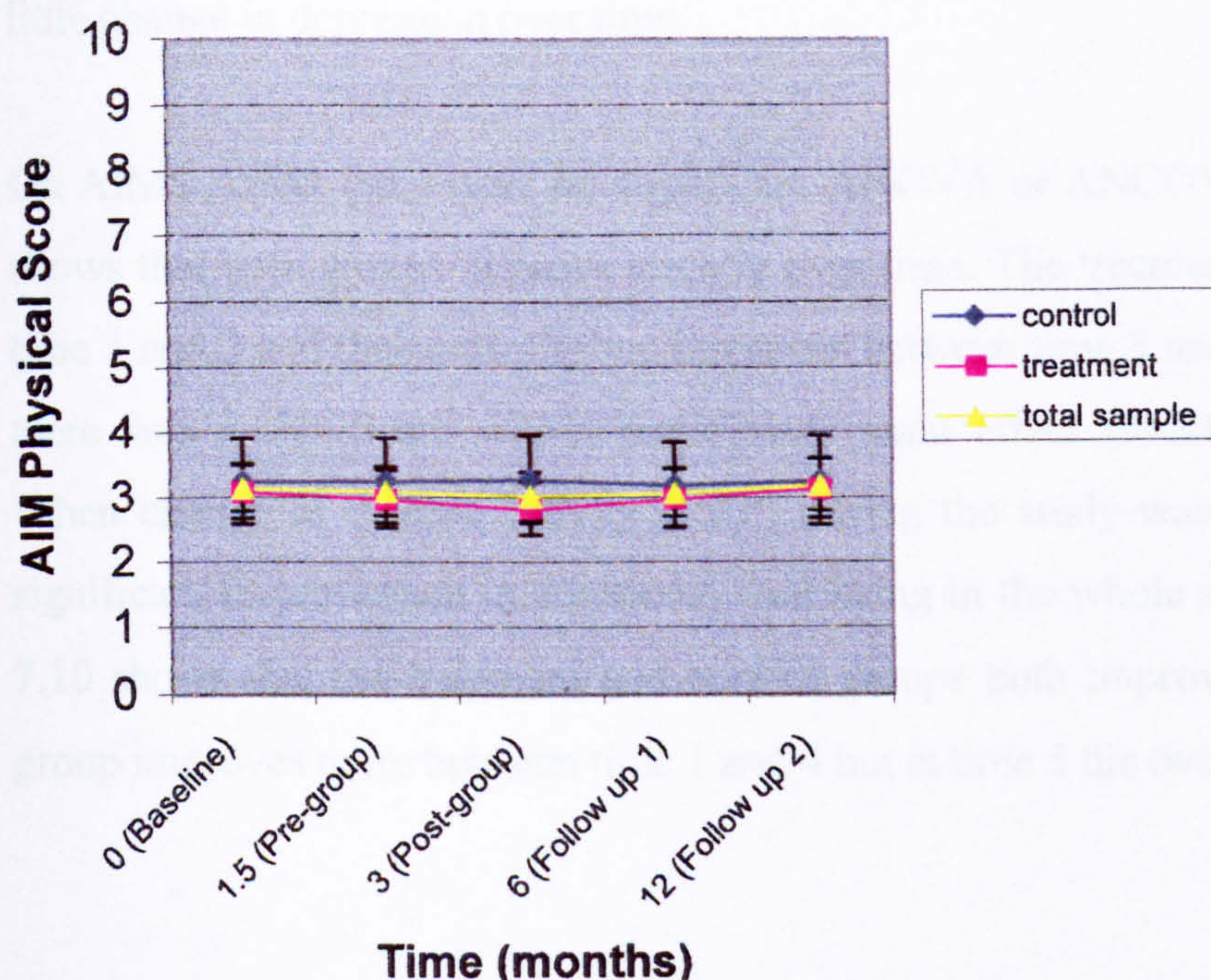
7.2(2) Emotional distress

On the HAQ's Anxiety scale there were no significant ANOVA or ANCOVA results therefore the hypothesis is rejected. Figure 7.5 shows that the treatment group's anxiety scores improve between time 1 and 4. There is a slight increase in anxiety at time 5. The control group's anxiety scores fluctuate over time but overall there is a slight increase in anxiety between time 1 and 5.

On the Hospital Depression Scale there were no significant ANOVA or ANCOVA results or interaction effects so the hypothesis is rejected. However on the Hospital Anxiety Scale at time 1 and 3 there was a significant within group main effect ($F_{(1, 11)} = 10.1, p = 0.005$) and a trend towards a significant interaction effect ($F_{(1, 11)} = 3.1, p = 0.07$).

Figure 7.9 shows that for the total sample there is a significant improvement in depression between time 1 and 3 but by time 4 and 5 the levels of depression return to original levels.

Figure 7.7: Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Physical scores between time 1 & 5



7.2(c) Emotional distress

On the HADS Anxiety scale there were no significant ANOVA or ANCOVA results therefore the hypothesis is rejected. Figure 7.8 shows that the treatment group's anxiety scores improve between time 1 and 4. There is a slight increase in anxiety at time 5. The control group's anxiety scores fluctuate over time but overall there is a slight increase in anxiety between time 1 and 5.

On the Hospital Depression Scale there were no significant ANOVA or ANCOVA main or interaction effects so the hypothesis is rejected. However on the planned contrasts for time 1 vs 3 there was a significant within participants main effect ($F = 5.567$, $df=1$, $p=0.020^*$) and a trend towards a significant interaction effect ($F=3.149$, $df=1$, $p=0.079$).

Figure 7.9 shows that for the total sample there is a small improvement in depression between time 1 and 3 but by time 4 and 5 the levels of depression return to slightly better

than baseline. Most of the change is accounted for by the treatment group which improves between time 1 and 3. There is a slight increase in depression at time 4, although it is still lower than baseline depression, this is maintained at time five. The control group shows little change in depression over time.

On AIMS Affect there were no significant ANOVA or ANCOVA (RI) results. Figure 7.10 shows that both groups improve slightly over time. The treatment group improves between time 1 and 3 and the control group improves between time 3 and 5. On the ANCOVA CRP there was a significant within participants main effect ($F=3.012$, $df=3.708$, $p= 0.021^*$). When change in disease activity (CRP) during the study was controlled for there was a significant improvement in emotional well being in the whole sample. Inspection of Figure 7.10 shows that the treatment and control groups both improve over time. The treatment group improves more between time 1 and 4 but at time 5 the two groups scores are identical.

Figure 7.8: Graph showing control, treatment & total sample mean & 95% confidence intervals for Hospital Anxiety scores between time 1 & 5

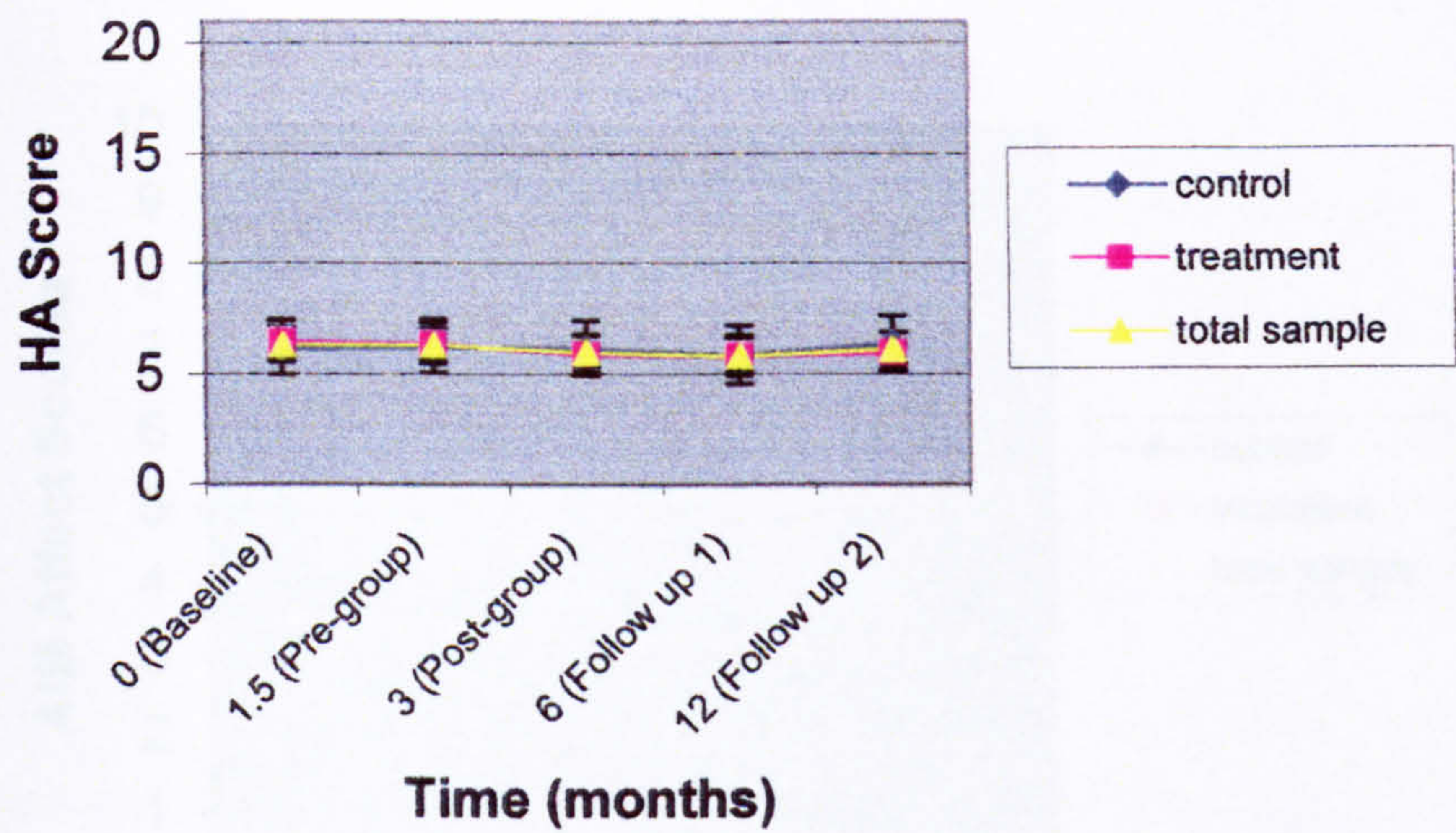
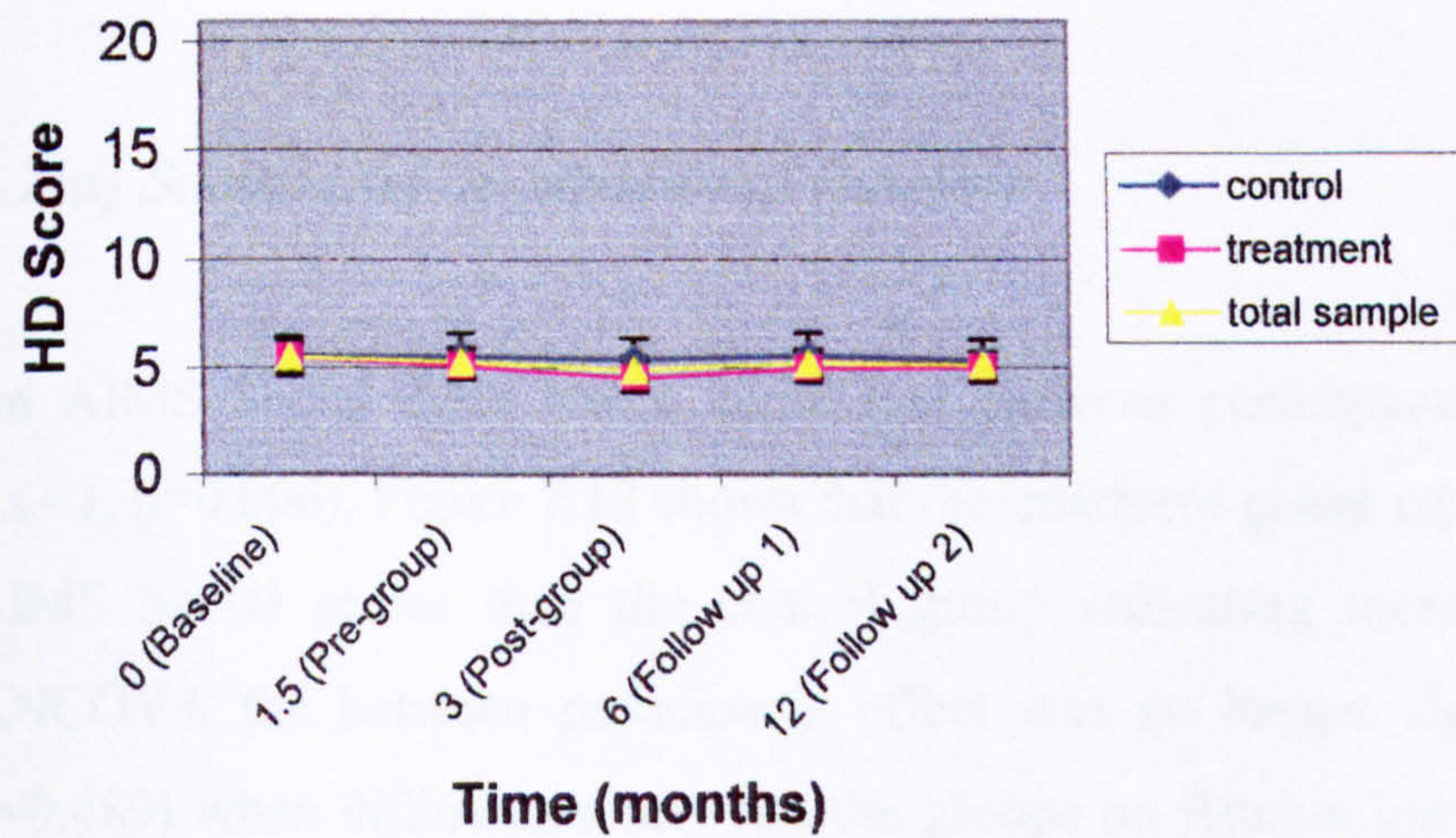
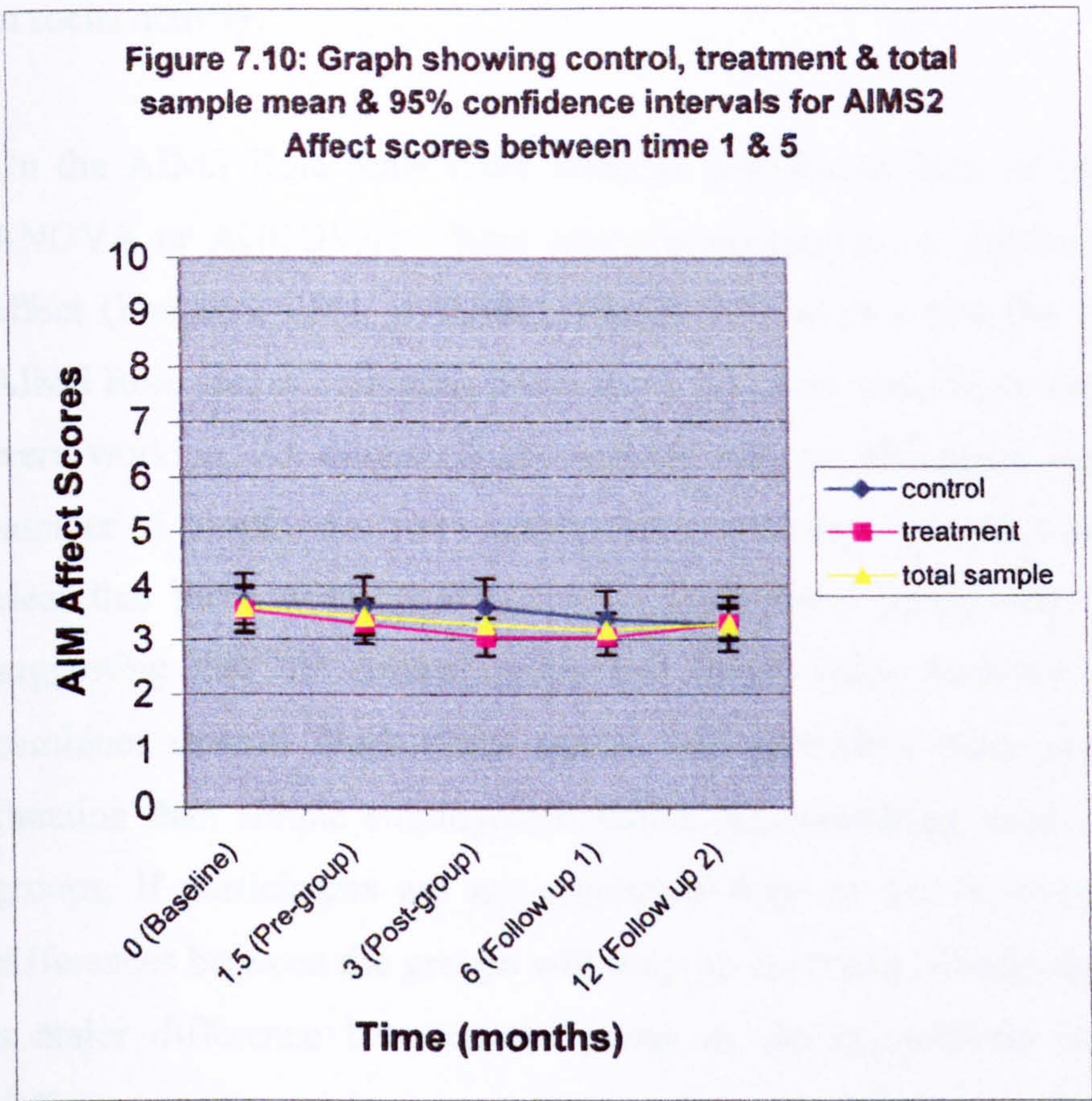


Figure 7.9: Graph showing control, treatment & total sample mean & 95% confidence intervals for Hospital Depression scores between time 1 & 5





7.2(d) Social and occupational function

On AIMS Social there was a significant between participants main effect ($F = 7.911$, $d.f.=1$, $p=0.006$). Figure 7.12 shows that the treatment group reported lower scores on the AIMS Social scales than the control group indicating more social activity. On the ANCOVA the between participants effect was no longer significant ($F=0.481$, $df=1$, $p=0.489$) when differences between the groups on Ritchie Index scores were controlled for.

On the planned contrasts there was a trend towards a significant interaction effect for time 1 vs 5 ($F=3.180$, $df=1$, $p=0.077$). Figure 7.12 shows that between time 1 and 5 the

control group report a decrease in social activity and the treatment group report an increase in social activity.

On the AIMS Role scale there were no significant main or interaction effects on the ANOVA or ANCOVAs. There was a trend towards a significant between participants effect ($F=3.066$, $df=1$, $p=0.084$). Figure 7.11 shows that the control group had lower AIMS Role scores indicating better work function than the treatment group for those who were working. As discussed above there was no difference between the groups in the number of people who were employed or non-employed. However from Table 6.17 it is clear that there were more people in the control group who were in full time work suggesting that the control group had better work function. The AIMS Role Scale combines several Work items and so will provide a more sensitive measure of work function than simple employment status, thus revealing subtle differences between the groups. If participants are not employed they do not complete this item and so any differences between the groups will only be for those completing the items. If there were a major difference between the group in the proportions completing this item any differences may not be representative of the whole sample. However the percentage of participants who responded to this item in each group is very similar, suggesting that the control group may have slightly better work function than the treatment group.

Figure 7.11 shows the whole sample had a decrease in AIMS Role score, which indicates an improvement in work function between time 1 and 5. This is confirmed by a significant within participants main effect on the planned contrast for time 1 vs 5 ($F=5.164$, $df=1$, $p=0.026^*$). Most of the improvement is in the control group. The control group shows a steady improvement over time. The treatment group improves between time 1 and 3 then deteriorates between time 3 and 4 before improving at time 5. Between time 3 and 4 the control group scores reduce and the treatment group scores increase which is supported by a trend towards a significant interaction effect for time 3 vs 4 ($F=3.4$, $df=1$, $p=0.07$).

Figure 7.11: Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Role scores between time 1 & 5

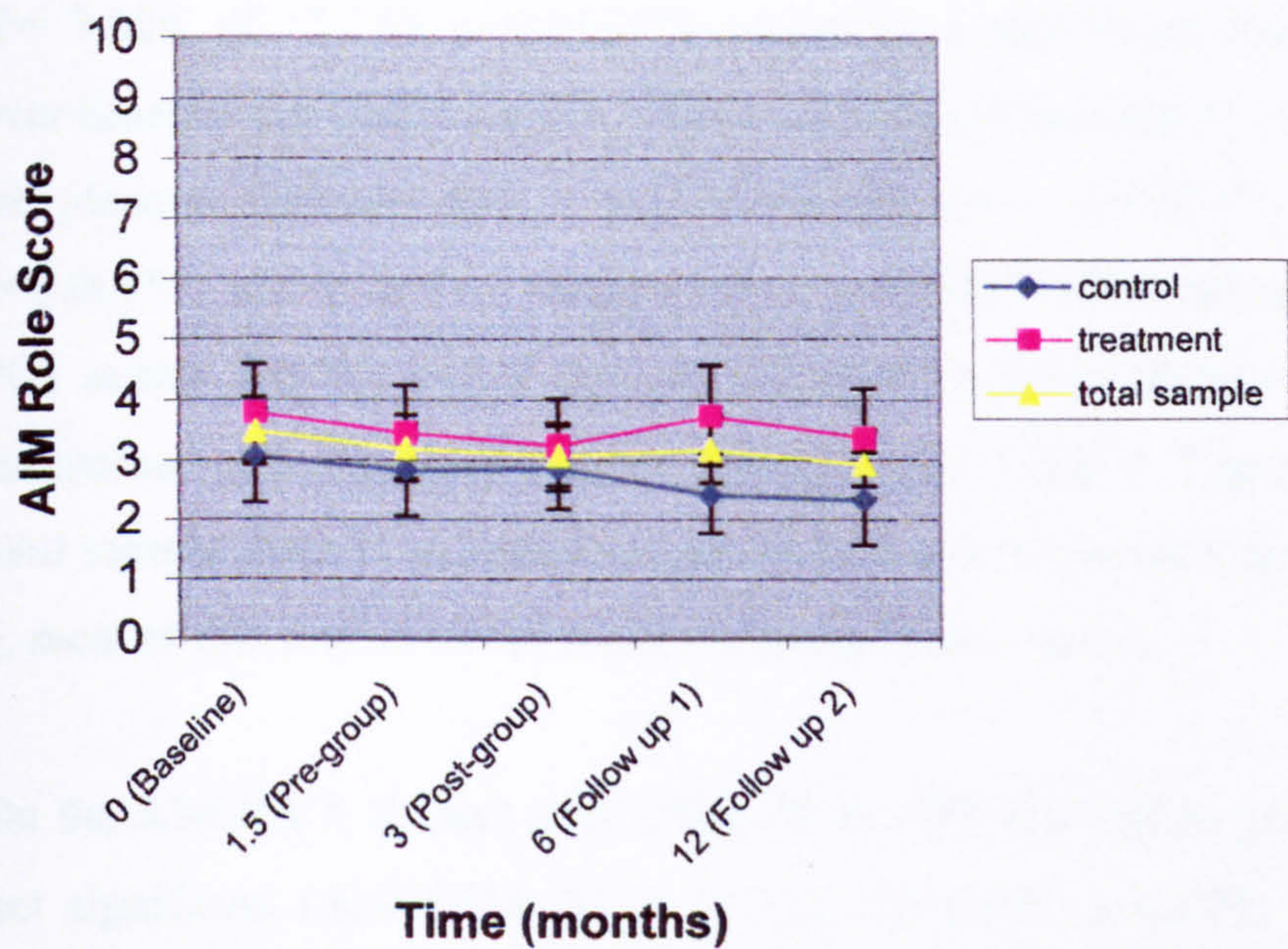
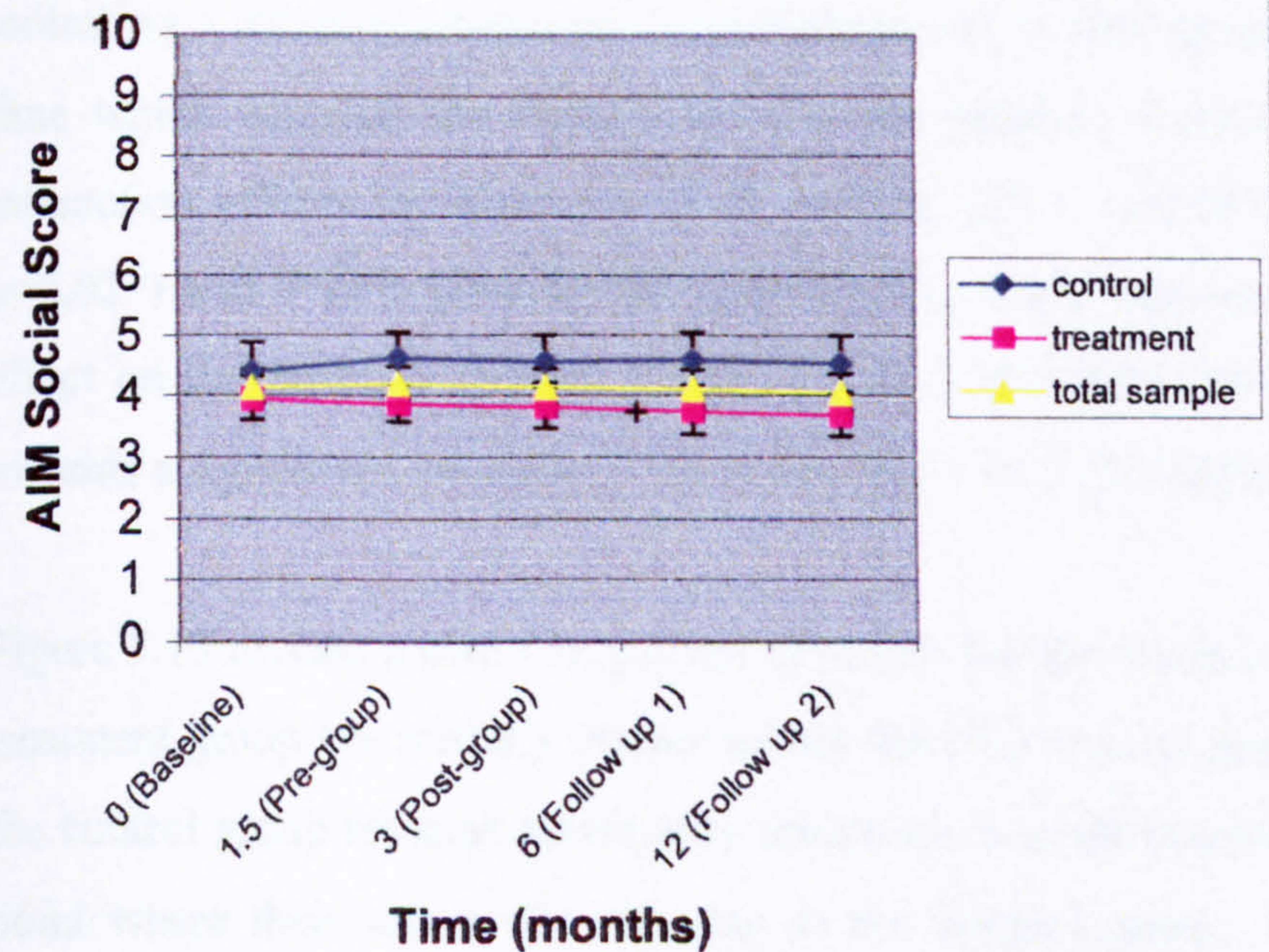


Figure 7.12: Graph showing control, treatment & total sample mean & 95% confidence intervals for AIMS2 Social scores between time 1 & 5



7.2(e) Individualized quality of life (PGI).

For the Patient Generated Index there was a significant within participants main effect ($F= 3.624$, $df =3.759$, $p=0.008^{**}$) indicating a significant improvement in PGI scores over time for the whole sample. There were significant within participant main effects for the planned contrasts time 1 vs 3 ($F=6.903$, $df=1$, $p=0.01^{**}$); 1 vs 4 ($F=7.025$, $df=1$, $p=0.009^{**}$) and 1 vs 5 ($F =8.210$, $df=1$, $p=0.005^{**}$) indicating that the improvement in PGI scores for the whole sample occurred mainly between time 1 and 3 and was maintained and improved slightly between time 3 and 5. Figure 7.13 shows that for the total sample there is an improvement in PGI scores between time 1 and times 2, 3, 4 and 5, most of this improvement occurs between time 1 and 3.

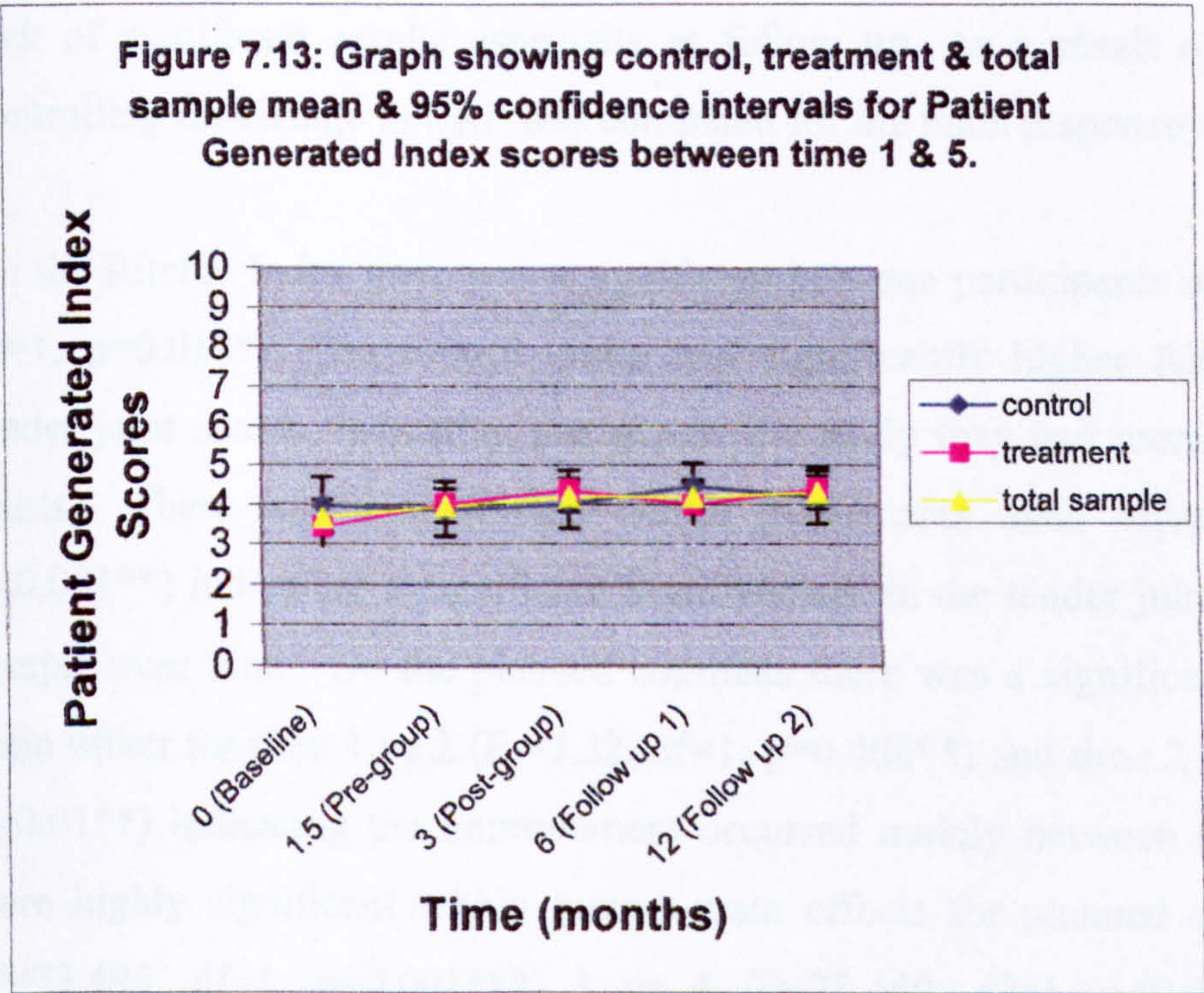
On the ANCOVA RI and ANCOVA RI & CRP the within participants main effect was not significant (ANCOVA RI $F =0.571$, $df=3.757$, $p=0.673$; ANCOVA RI & CRP $F =0.643$, $df=3.789$, $p =0.623$) suggesting that the within participants main effect could be accounted for by the difference between the groups in Ritchie Index scores.

On the PGI there was a significant interaction effect ($F=2.674$, $df=3.759$, $p=0.035^{*}$) indicating a difference between the treatment and control group in their PGI scores over time which supports the hypothesis. On the planned contrasts there were significant interaction effects for time 1vs 2 ($F =3.913$, $df=1$, $p=0.05^{*}$); 3 vs 4 ($F=5.97$, $df=1$, $p=0.02^{*}$) and 4 vs 5 ($F=4.03$, $df=1$, $p=0.05^{*}$). There was also a significant interaction effect on the planned contrast for time 1 vs 3 ($F=6.053$, $df=1$, $p=0.015^{*}$) and a trend towards a significant interaction effect for time 1 vs 5 ($F=3.675$, $df=1$, $p=0.058$).

Figure 7.13 reveals a complex pattern of scores for the control and treatment groups. The treatment group has initially poorer scores than the control group. Between time 1 and 3 the control group remains reasonably constant while the treatment group improves to the point where their scores are superior to the control group. At the first follow up the control group improves and the treatment group declines so that the control group's scores are superior. At the second follow up at time 5, the situation is reversed and the

treatment group improves and the control group declines slightly. Overall, during the study the control group's score increases slightly from time 1 to 5 and the treatment group's score improves significantly.

Effect sizes (Cohen d) were calculated separately for the treatment and control groups' change in individualized quality of life between time 1 and 5 ([mean time 5-time 1]/[pooled standard deviation time 5 and time 1]). The treatment group had a small to moderate positive effect size, 0.45, and the control group had a very small positive effect size, 0.08 (Kazis, Anderson & Meenan, 1989).



7.2(f) Disease activity

Overall there was a reduction in disease activity measured on the CRP for the whole sample across the course of the study, confirmed by a significant within participants main effect ($F = 3.708$, $d.f. = 3.087$, $p = 0.011^*$). The planned contrasts show a significant within participants main effect between time 1 and 2 ($F = 6.69$, $d.f. = 1$, $p = 0.01$). Planned

contrasts showed a trend towards a significant reduction in CRP for the whole sample from time 1 vs 3 ($F=3.831$, $df=1$, $p=0.053$), and a significant reduction between time 1 vs 4 ($F=5.596$, $df=1$, $p=0.026^*$) and time 1 vs 5 ($F=8.739$, $df=1$, $p=0.004^{**}$).

Inspection of Figure 7.15 shows that the control group had higher CRP scores at the start of the study. Both groups show a reduction in scores over time but the control group shows the largest reduction. The treatment group shows a steady reduction over time. The control group reduces rapidly between time 1 and 2, has a slight increase at time 3 and then reduces again at time 4 and 5.

It is possible that these improvements in the acute phase response might account for the lack of significant results especially at follow up. As a result of this the ANCOVA controlling for change in CRP was computed for the main response variables.

On the Ritchie Index there was a significant between participants main effect ($F=5.732$, $df=1$, $p=0.018^*$). The control group had significantly higher Ritchie Articular Index tender joint scores, indicating that across the study they had more swollen and painful joints. There was a significant within participants main effect ($F=13.232$, $df=4$, $p<0.001^{**}$) indicating a significant improvement in the tender joint score for the whole sample over time. On the planned contrasts there was a significant within participants main effect for time 1 vs 2 ($F=7.32$, $df=1$, $p=0.008^{**}$) and time 2 vs 3 ($F=6.198$, $df=1$, $p=0.01^{**}$) indicating the improvement occurred mainly between time 1 and 3. There were highly significant within groups main effects for planned contrasts time 1 vs 3 ($F=23.496$, $df=1$, $p<0.001^{***}$) 1 vs 4 ($F=21.460$, $df=1$, $p<0.001^{***}$) and 1 vs 5 ($F=29.792$, $df=1$, $p<0.001^{***}$). These results show that there was a significant improvement by time 3, which was maintained and increased over time. In summary the whole sample showed improvement in their Ritchie Index scores during the study. This pattern of results can be seen in Figure 7.16 which shows that the control group had higher Ritchie scores (more tender joints) throughout the study. Both groups improved over time especially in the first three months and the control group had larger changes.

For EMS there were no significant main or interaction effects on the ANOVA or ANCOVA. On the planned contrasts there was a significant within participants main effect for time 1 vs 3 ($F=5.559$, $df=1$, $p=0.02^*$). Figure 7.14 shows a marked improvement in both groups between time 1 and 3. There was a significant interaction effect for time 4 vs 5 ($F=5.2$, $df=1$, $p=0.02^*$). The Treatment group continues improving at time 4 but then shows an increase in EMS to slightly below baseline at time 5. The control group shows an increase in EMS at time 4 and then a marked reduction at time 5. The deterioration in the treatment group and the improvement in the control group between time 4 and 5 explains the significant interaction effect and may indicate a difference in disease activity between the groups during the follow up period supporting the possibility that this may have interfered with maintenance of gains from the intervention.

For Hb there was a significant within participant main effect ($F=7.155$, $df=1.803$, $p=0.001^{***}$). This was not significant on the ANCOVA ($F=1.573$, $df=1.780$, $p=0.212$). On the planned contrasts there were significant within participants effects for time 1 vs 4 ($f=8.794$, $df=1$, $p=0.004^{**}$) and time 1 vs 5 ($F=9.950$, $df=1$, $p=0.002^{**}$). Figure 7.19 shows that both groups improve (increased Hb levels) over time with most improvement between time 1 and 4.

There were no significant results on the ANOVA or ANCOVA for White Cell Count, Platelets or ESR.

Figure 7.14: Graph showing control, treatment & total sample mean & 95% confidence intervals for Early Morning Stiffness scores between time 1 and 5

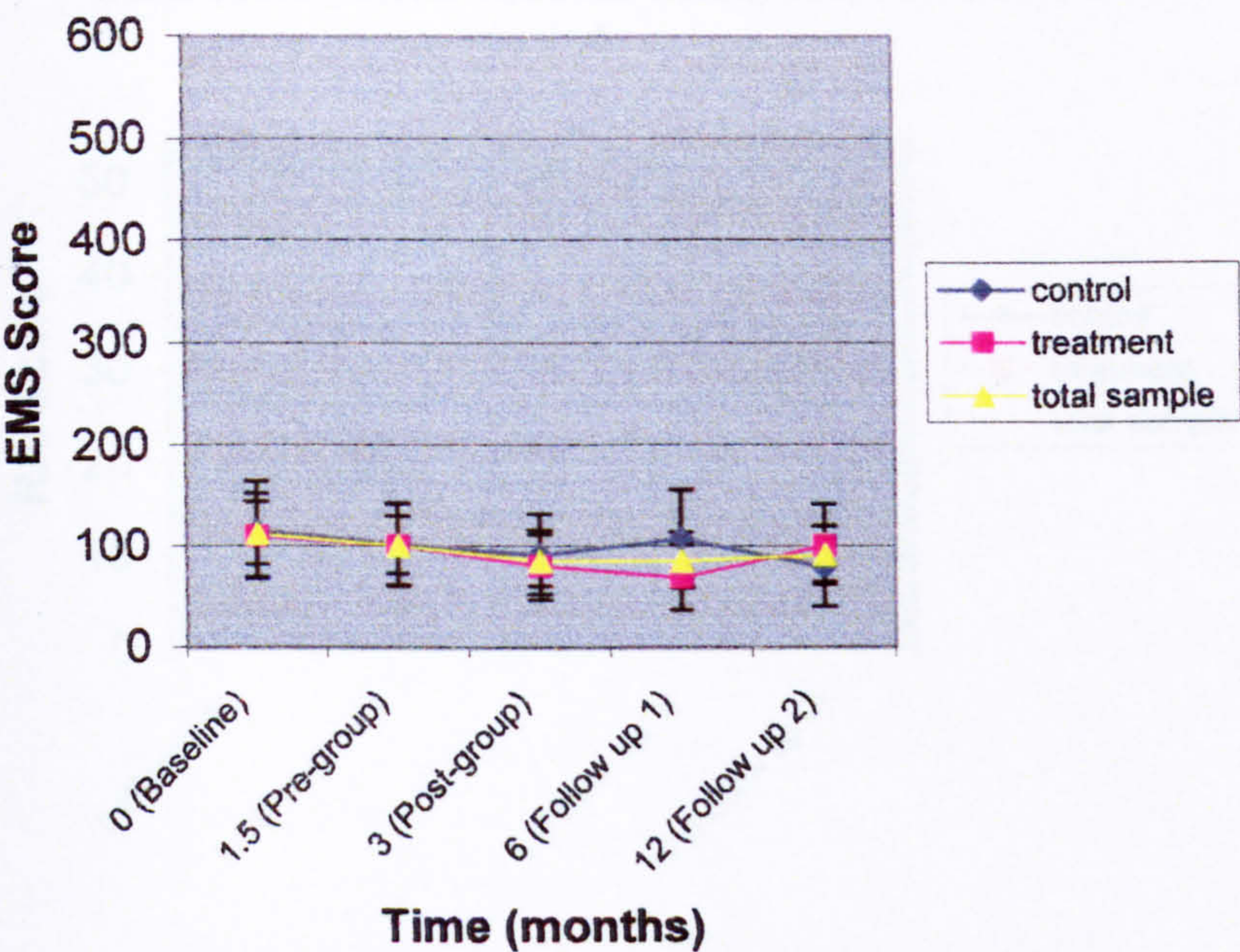


Figure 7.15: Graph showing control, treatment & total sample mean & 95% confidence intervals for CRP scores between time 1 & 5

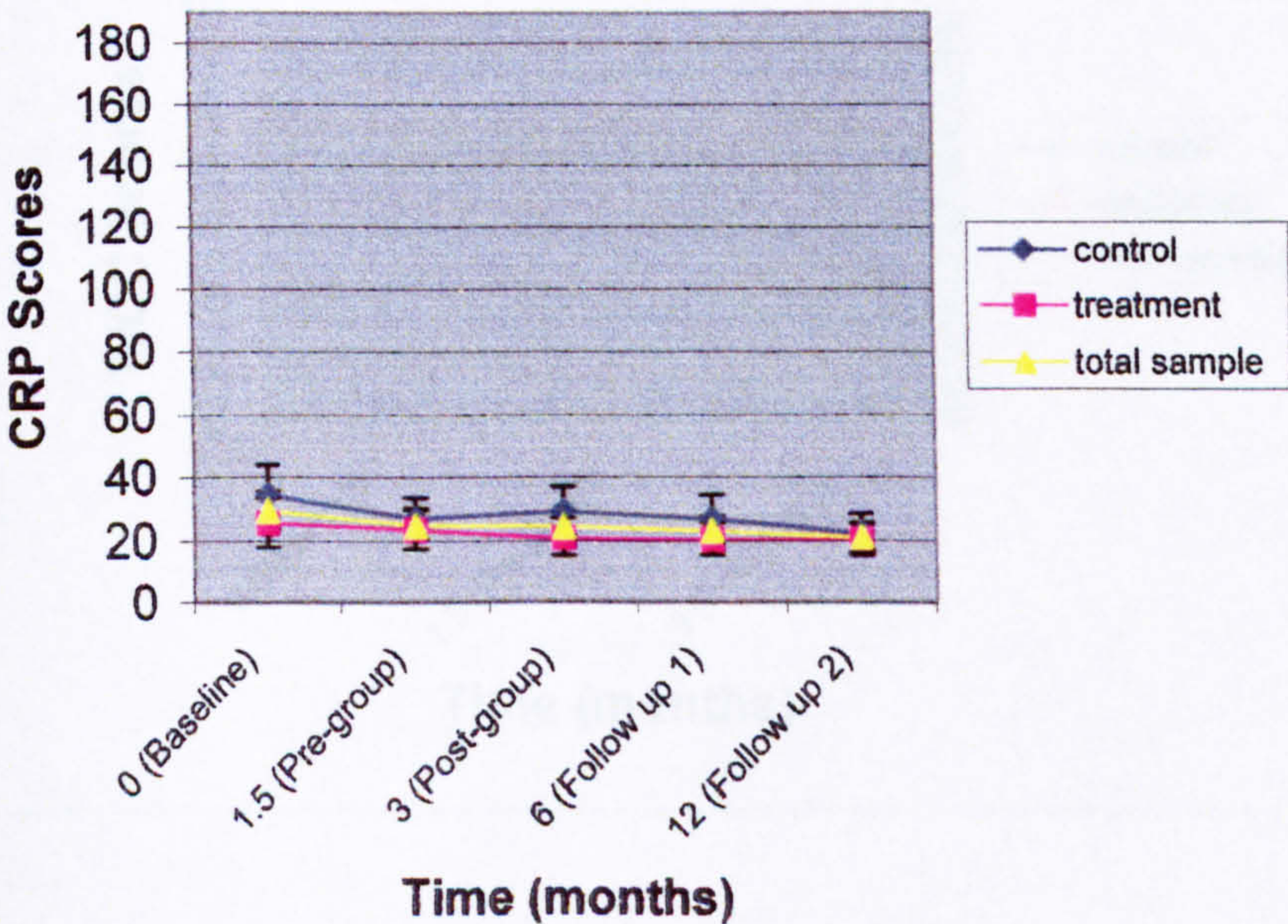


Figure 7.16: Graph showing control, treatment & total sample mean & 95% confidence intervals for Tender Joint Score (Ritchie Articular Index) between time 1 & 5

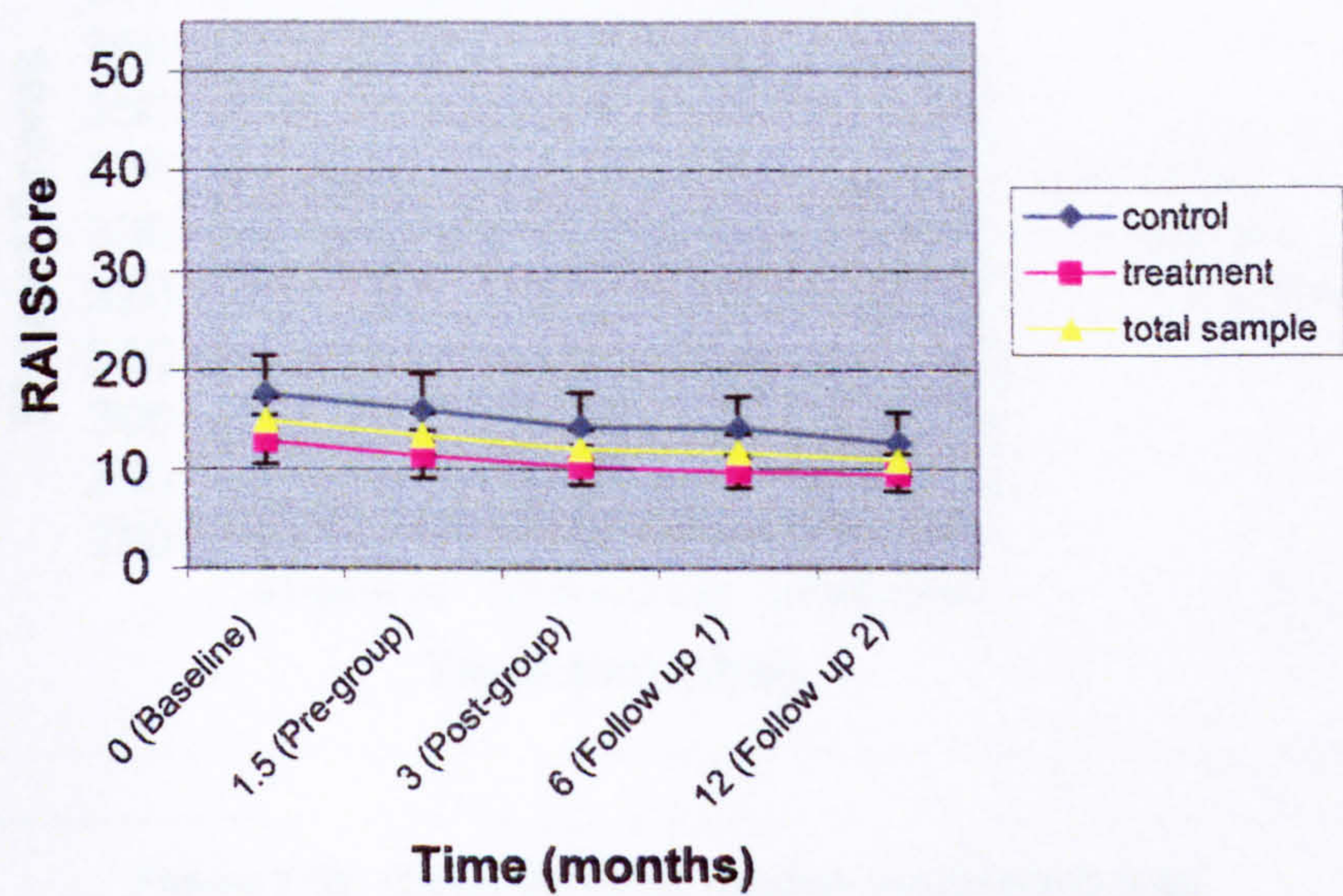


Figure 7.17: Graph showing control, treatment & total sample mean & 95% confidence intervals for WCC scores between time 1 & 5.

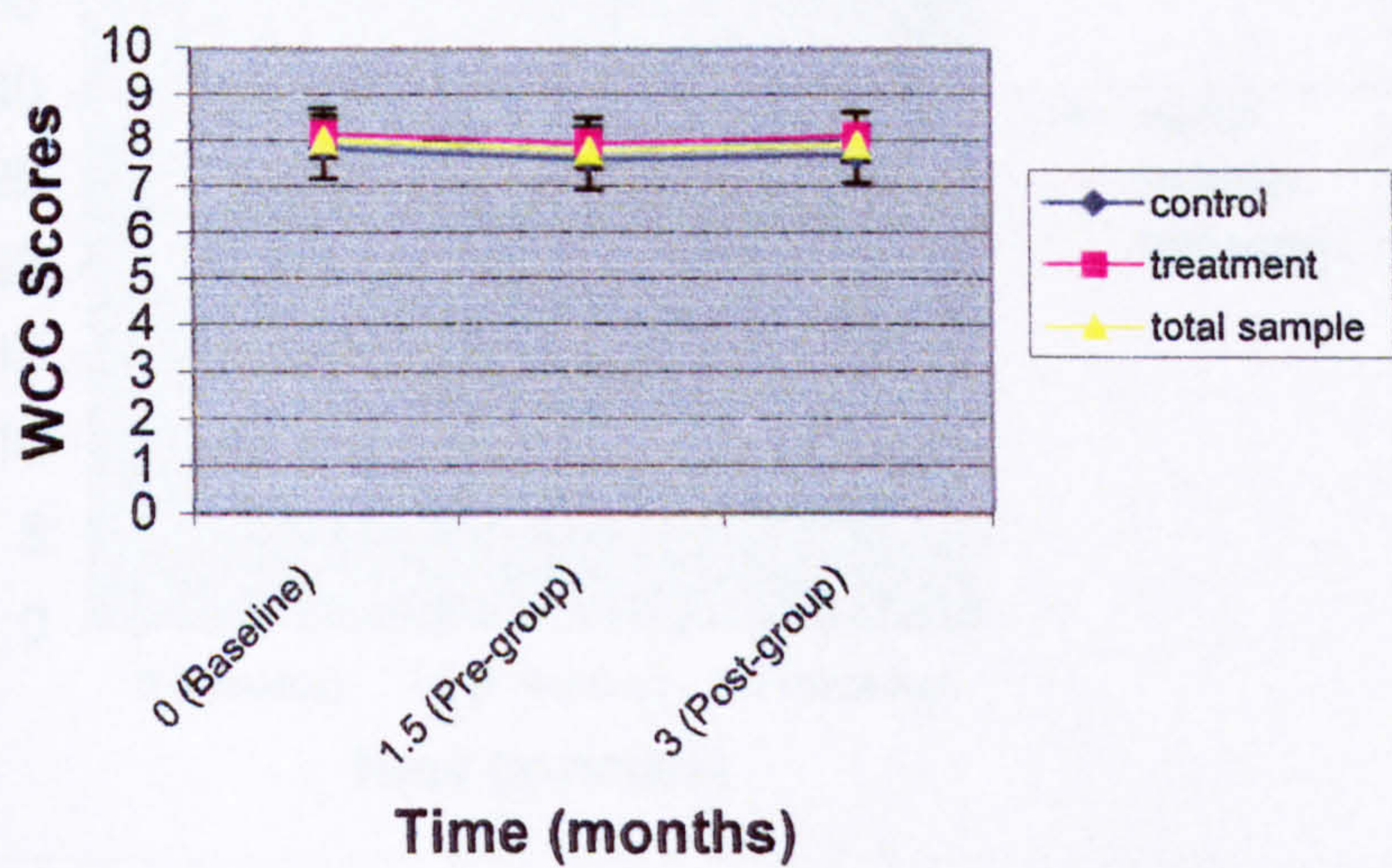


Figure 7.18: Graph showing control, treatment & total sample mean & 95% confidence intervals for Platelet scores between time 1 & 5

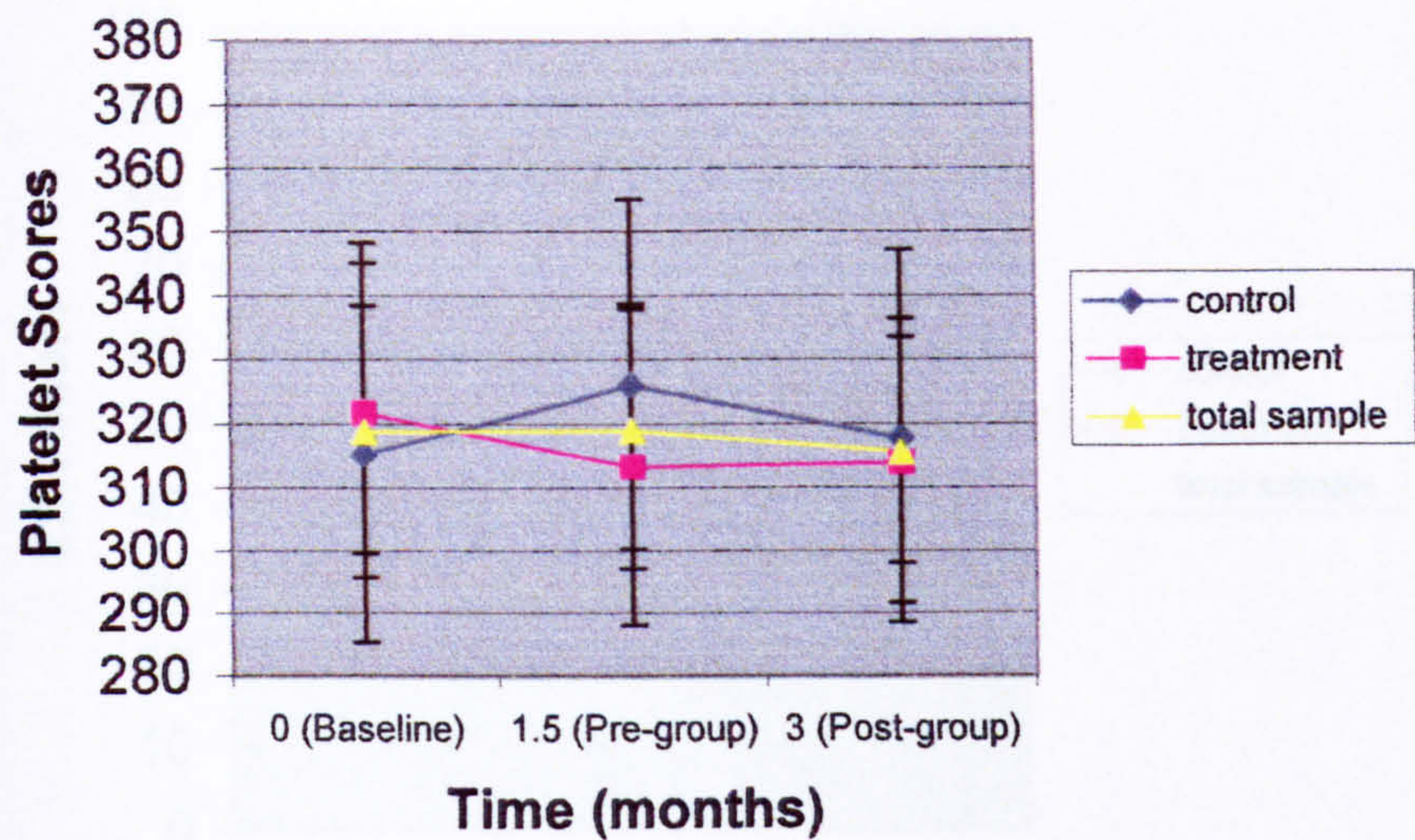


Figure 7.19: Graph showing control, treatment & total sample mean & 95% confidence intervals for HB scores between time 1 & 5

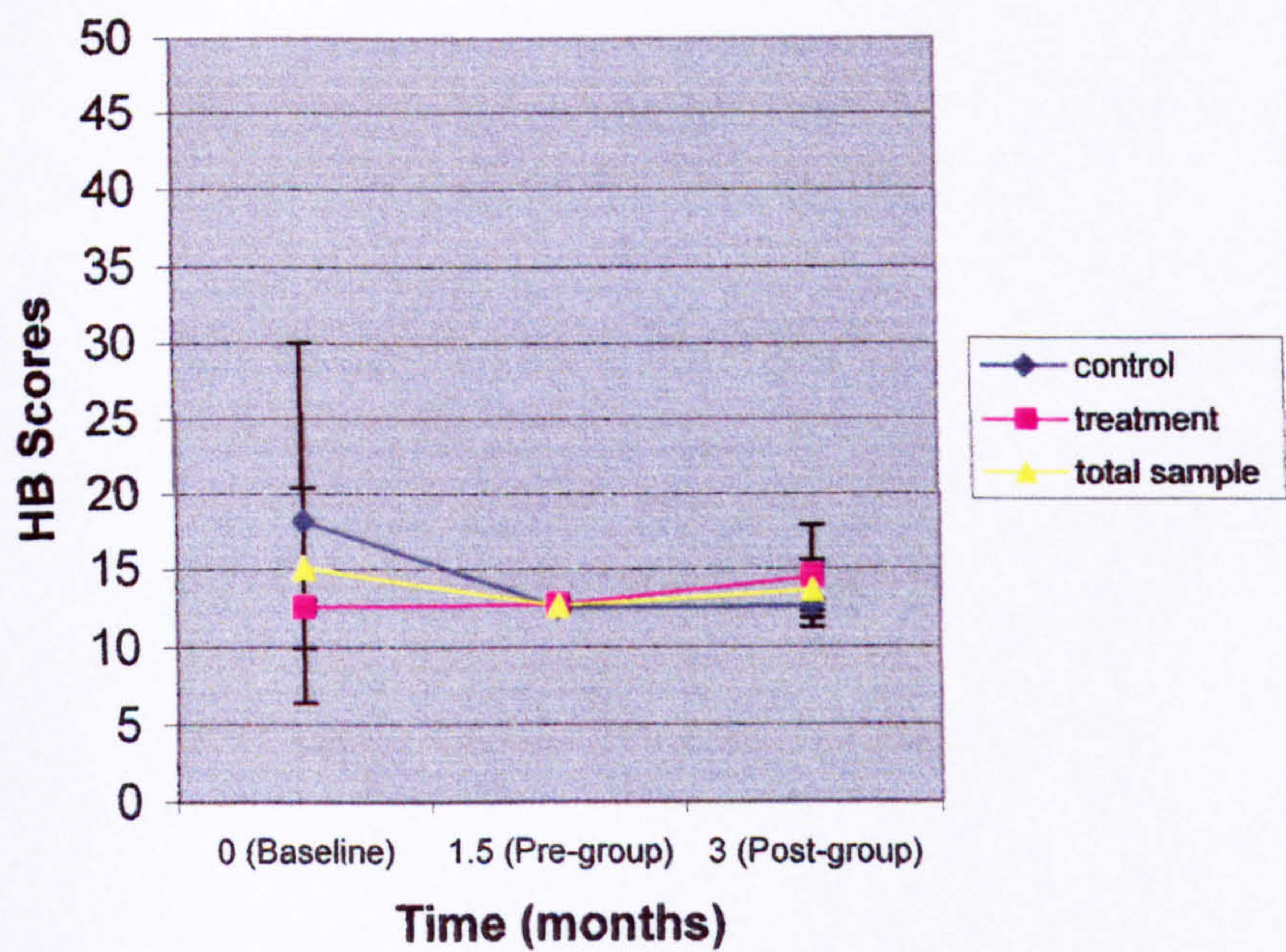
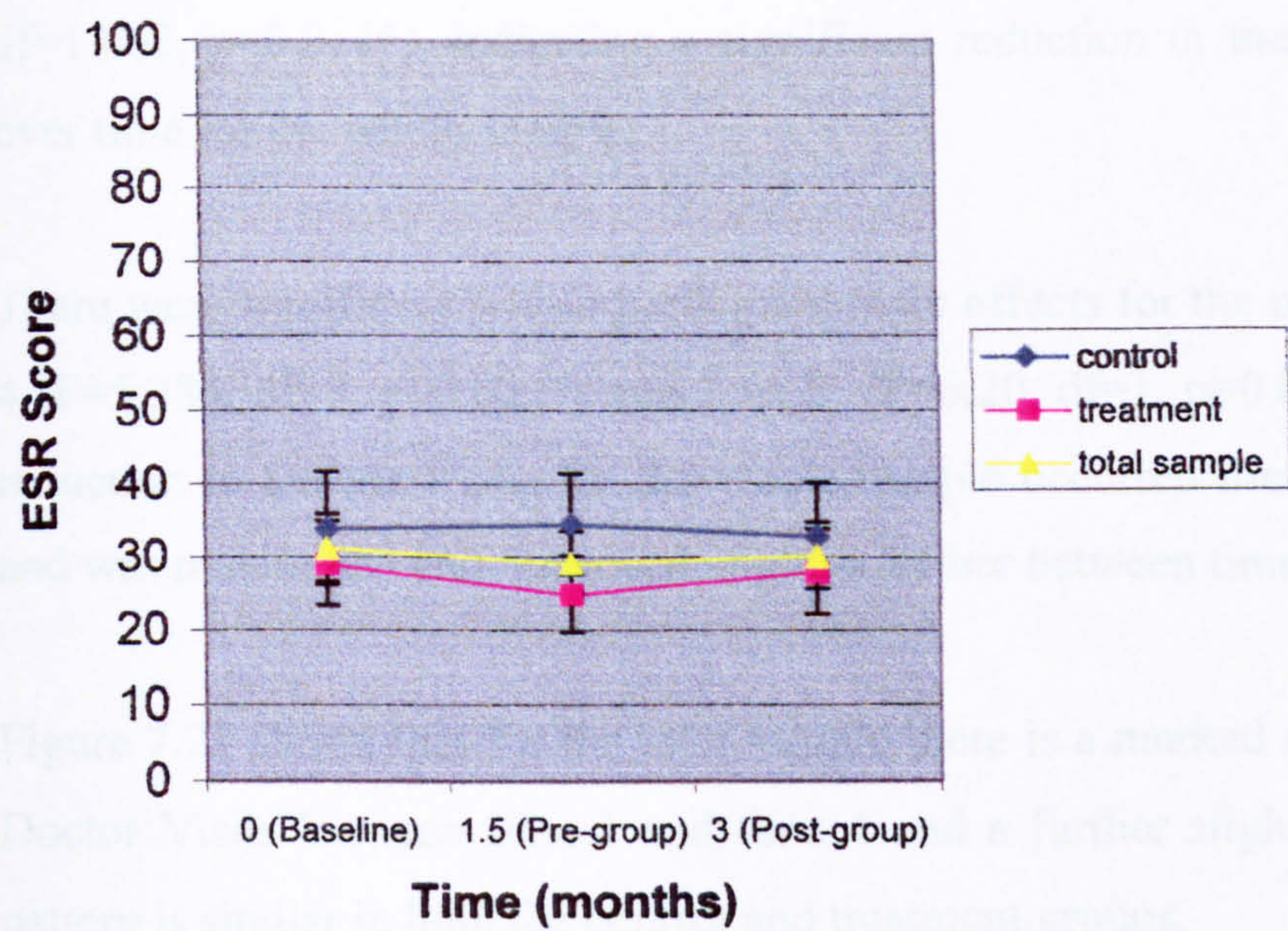


Figure 7.20: Graph showing control, treatment & total sample mean & 95% confidence intervals for ESR scores between time 1 & 5



7.2(g) *Health care utilization*

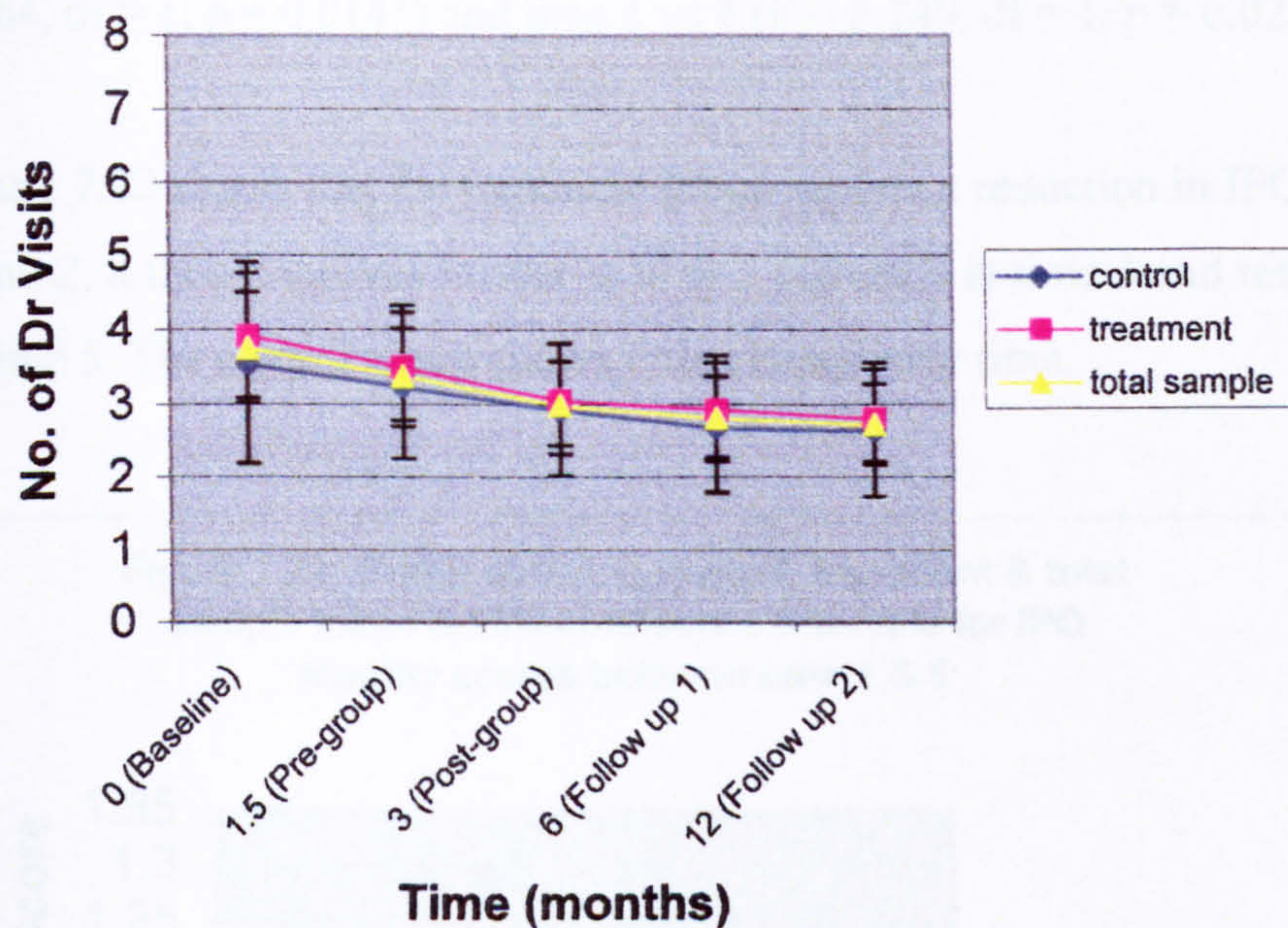
For Doctor Visits there was a significant within participants main effect ($F = 5.049$, $df=1.462$, $p=0.014^*$), indicating a significant reduction in the number of Doctor Visits over time for the whole sample.

There were significant within participant main effects for the planned contrasts time 1 vs 4 ($F=5.384$, $df=1$, $p=0.022^*$) and 1 vs 5 ($F=6.20$, $df=1$, $p=0.014^*$). This shows that the reduction in Doctor Visits for the whole sample occurred mainly between time 1 and 4 and was maintained and improved slightly further between time 4 and 5.

Figure 7.21 shows that for the total sample there is a marked reduction in the number of Doctor Visits between time 1 and time 4 and a further slight reduction at time 5. The pattern is similar in both the control and treatment groups.

On the ANCOVA RI and ANCOVA RI & CRP the within participants main effect was not significant (ANCOVA RI $F=2.169$, $df=1.464$, $p=0.132$; ANCOVA RI & CRP $F=2.173$, $df=1.466$, $p=0.131$), suggesting that the within participants main effect could be accounted for by the time 1 Ritchie Index scores.

Figure 7.21: Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of Dr Visits between time 1 & 5



7.3 Changes in illness representations and coping procedures following self-management intervention

The second set of analyses addressed Research Question 1.5 And Hypothesis 1.2

RQ1.5 How do illness representations and coping procedures in a sample of people with RA change over one year?

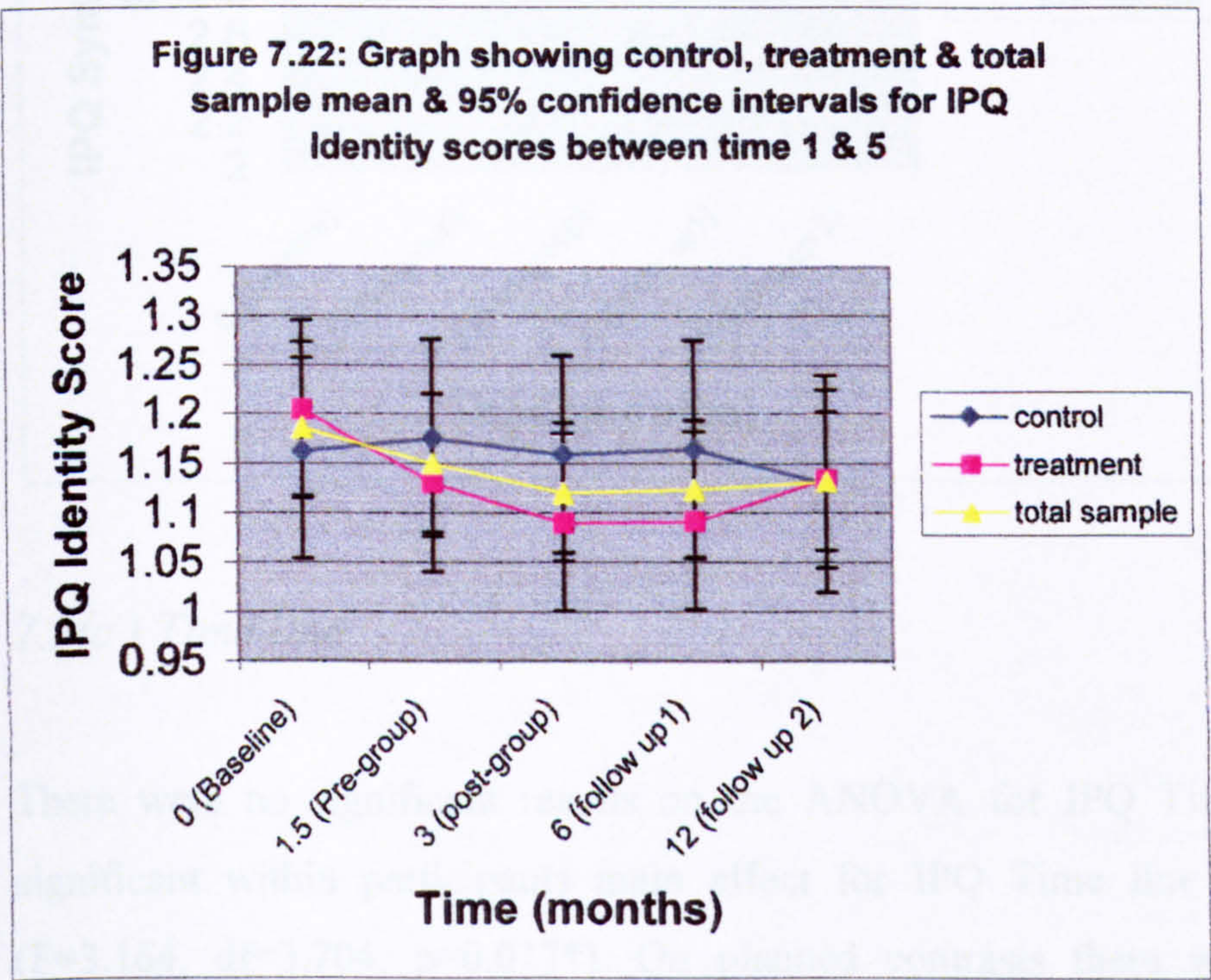
Hypothesis 1.2 The intervention will not produce any significant change in the following illness representations and coping strategies.

7.3(a) Illness identity

For IPQ Identity There were no significant results on the main ANOVA or ANCOVA.

On the planned contrasts there was a significant within participants main effect for time 1 vs 3 ($F = 6.91, df = 1, p = 0.010^{**}$) and time 1 vs 4 ($F = 4.860, df = 1, p = 0.029^{*}$). There was a significant interaction effect for time 1 vs 2 ($F = 4.36, df = 1, p = 0.04^{*}$), time 1 vs 3 ($F = 6.164, df = 1, p = 0.014^{*}$) and time 1 vs 4 ($F = 5.149, df = 1, p = 0.025^{*}$).

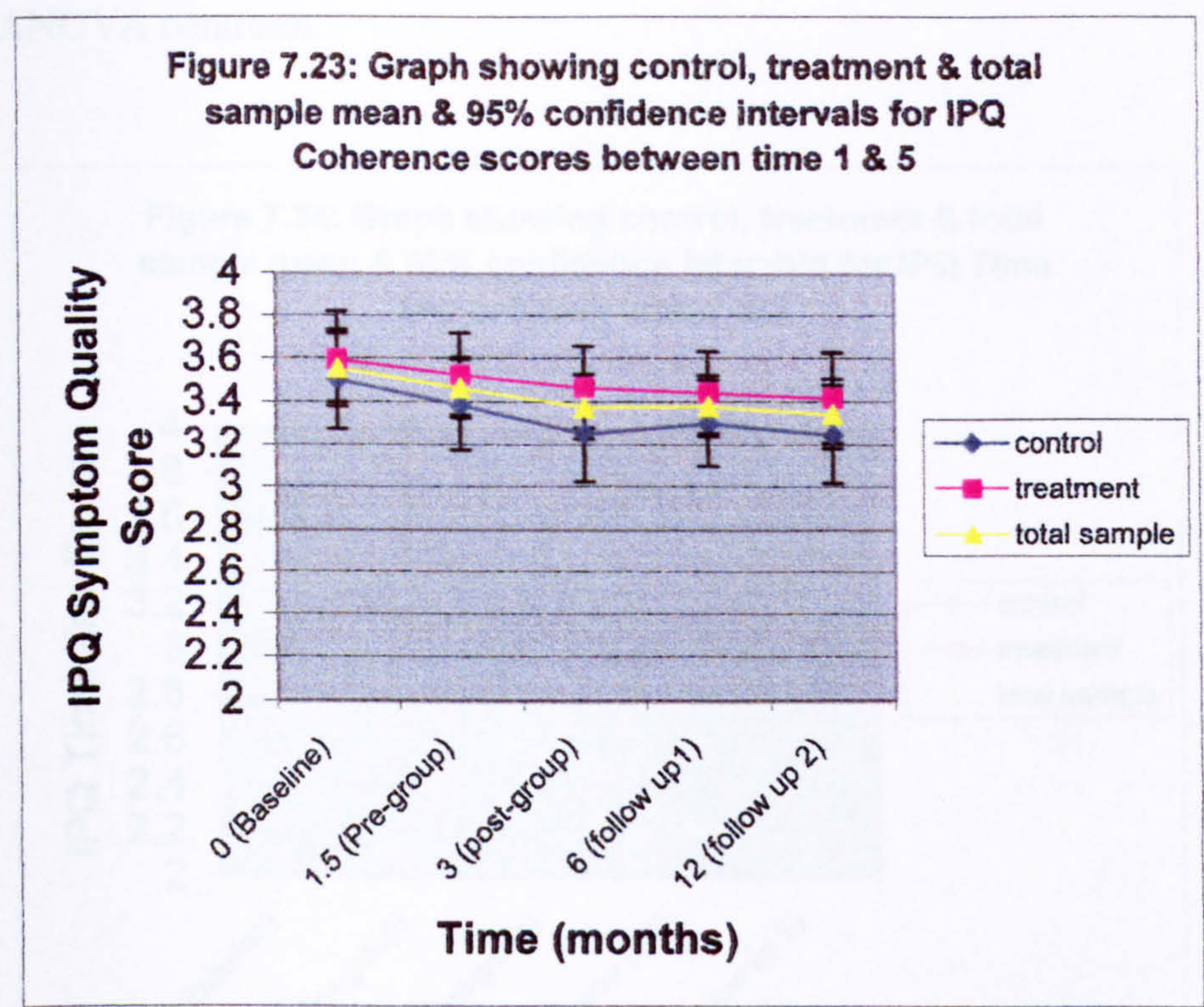
Figure 7.22 shows that the treatment group reports a reduction in IPQ Identity between time 1 and 2, it then improves further at time 3 plateau's at time 4 and returns to the time 2 level at time 5. The control group shows little change over time.



7.3(b) Symptom coherence

There was a significant within participants main effect for IPQ Symptom Coherence ($F = 4.269, df = 3.260, p = 0.004^{**}$). On the planned contrasts there were significant within participants effects for time 1 vs 3 ($F = 8.398, df = 1, p = 0.004^{**}$) 1 vs 4 ($F = 7.225, df = 1, p = 0.008^{**}$) and 1 vs 5 ($F = 8.022, df = 1, p = 0.005^{**}$).

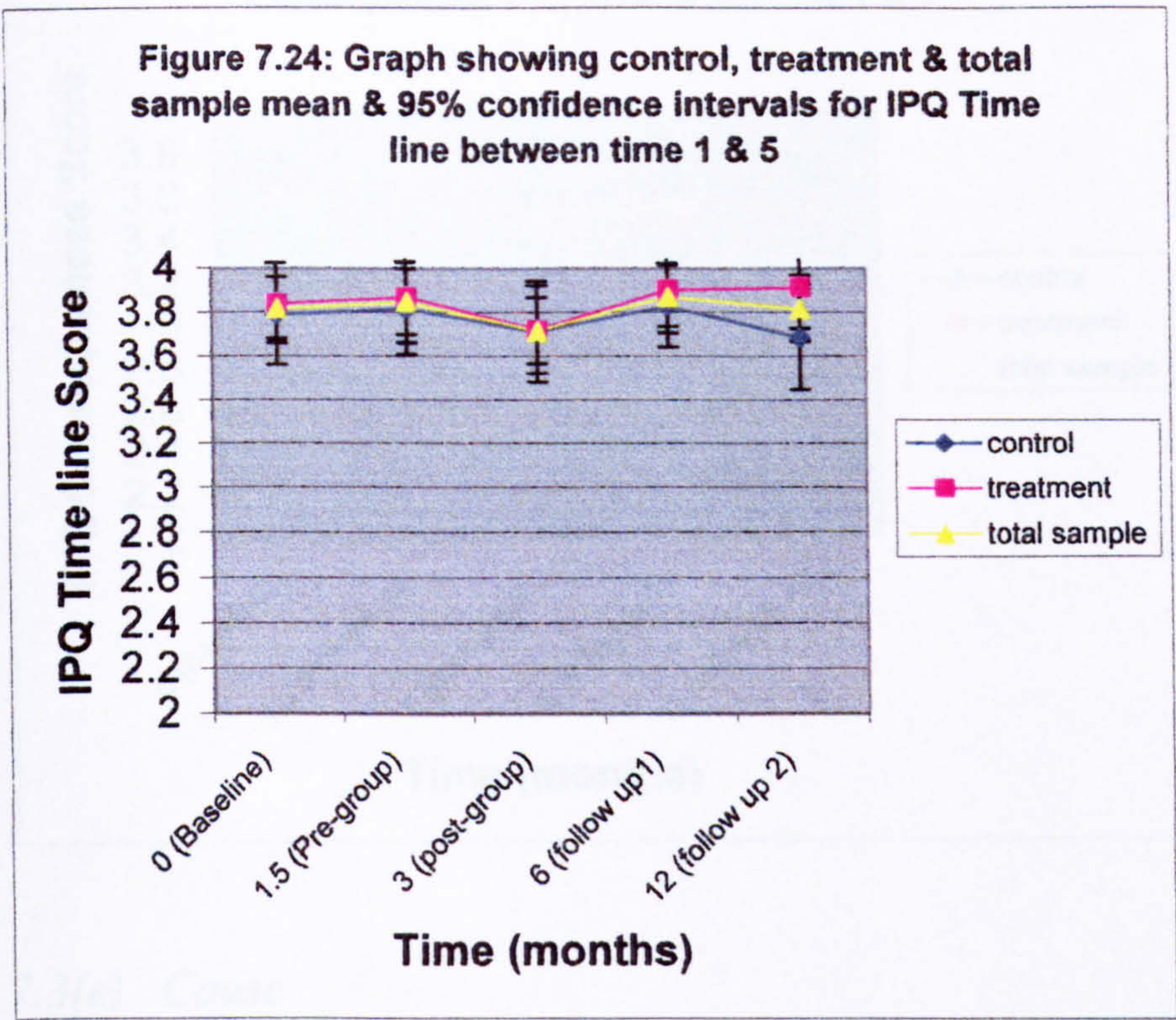
Figure 7.23 shows that both groups report improved symptom coherence between time 1 and 3. The improvement is maintained at time 4 and 5.



7.3(c) Time line

There were no significant results on the ANOVA for IPQ Time Line. There was a significant within participants main effect for IPQ Time line on the ANCOVA RI ($F=3.164$, $df=3.704$, $p=0.017^*$). On planned contrasts there were significant within participants effects for time 2 vs 3 ($F = 5.77$, $df = 1$, $p = 0.02^*$) and 3 vs 4 ($F = 6.81$, $df = 1$, $p = 0.01^{**}$). On Figure 7.24 at time 1 the treatment group has a slightly more chronic time line perception than the control group. Both groups show a decrease in the chronicity of their timeline perception between time 2 and 3 and an increase between time 3 and 4. Between time 4 and 5 the treatment group maintains the increase in the chronicity of their time line perception and the control group shows a reduction. At time 5 the treatment group have a more chronic time line perception than the control group. However, for the whole sample there is little difference between time 1 and 5. This significant within participant effect is difficult to interpret. Therefore, ANCOVA RI contrasts were computed comparing

time 1 vs 2, 2 vs 3, 3 vs 4 and 4 vs 5, 1 vs 3, 1 vs 4 and 1 vs 5 controlling for initial Ritchie Index scores. These did not reveal any differences in the patterns of results from the ANOVA contrasts.

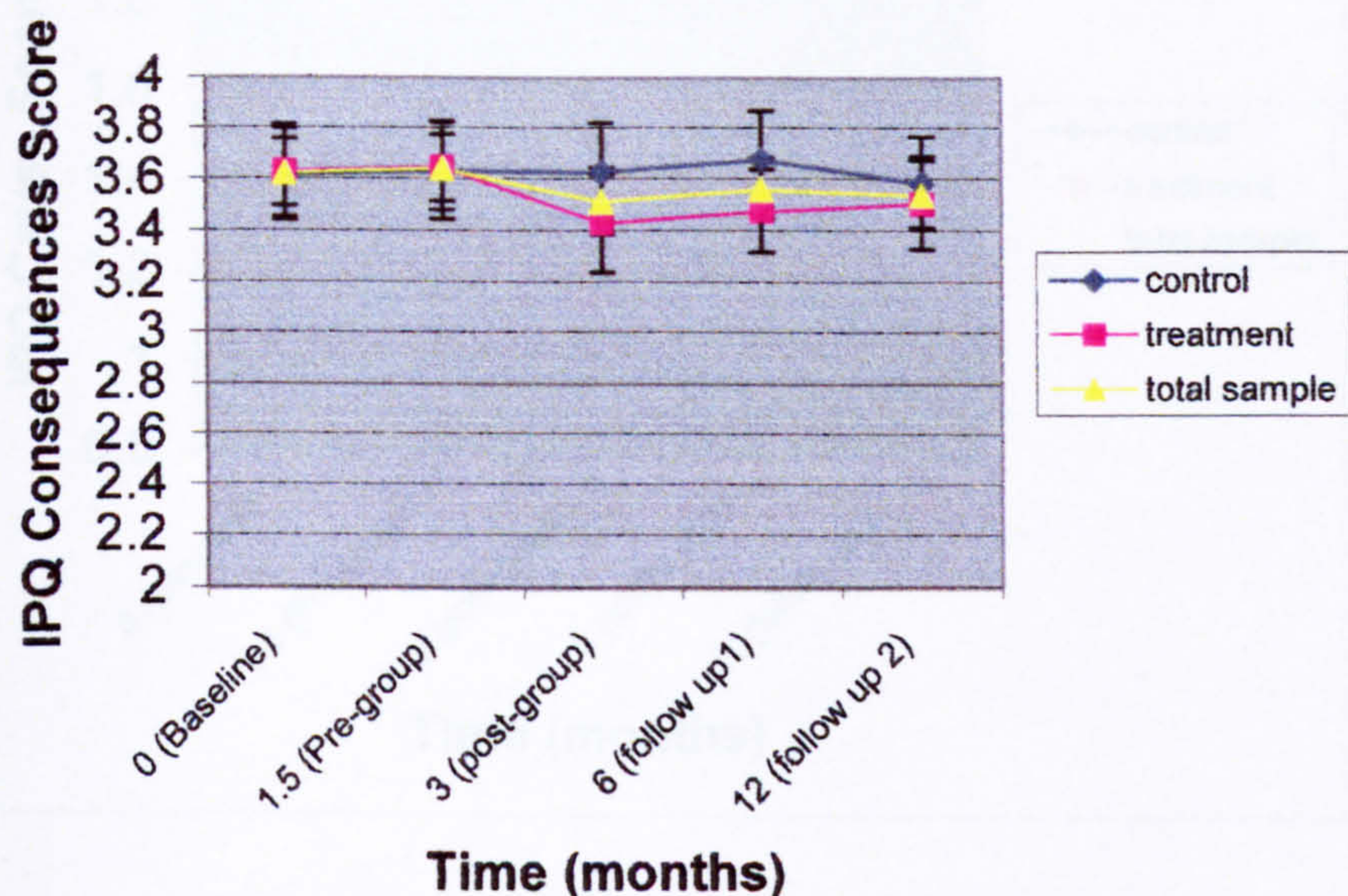


7.3(d) Consequences

For IPQ Consequences there were no significant results on the main ANOVAs or ANCOVAs. On planned contrasts there was a significant within participants effect for time 2 vs 3 ($F = 4.76$, $df = 1$, $p = 0.03^*$). There was a significant interaction effect for time 2 vs 3 ($F = 4.35$, $df = 1$, $p = 0.04^*$) and time 1 vs 3 ($F = 3.918$, $df = 1$, $p = 0.05^*$).

On Figure 7.25 the treatment group show a decrease in the perceived consequences of RA between time 2 and 3, following the intervention. However by time 5 they perceived the consequences of RA to be only slightly less than at time 1. The control group show little change.

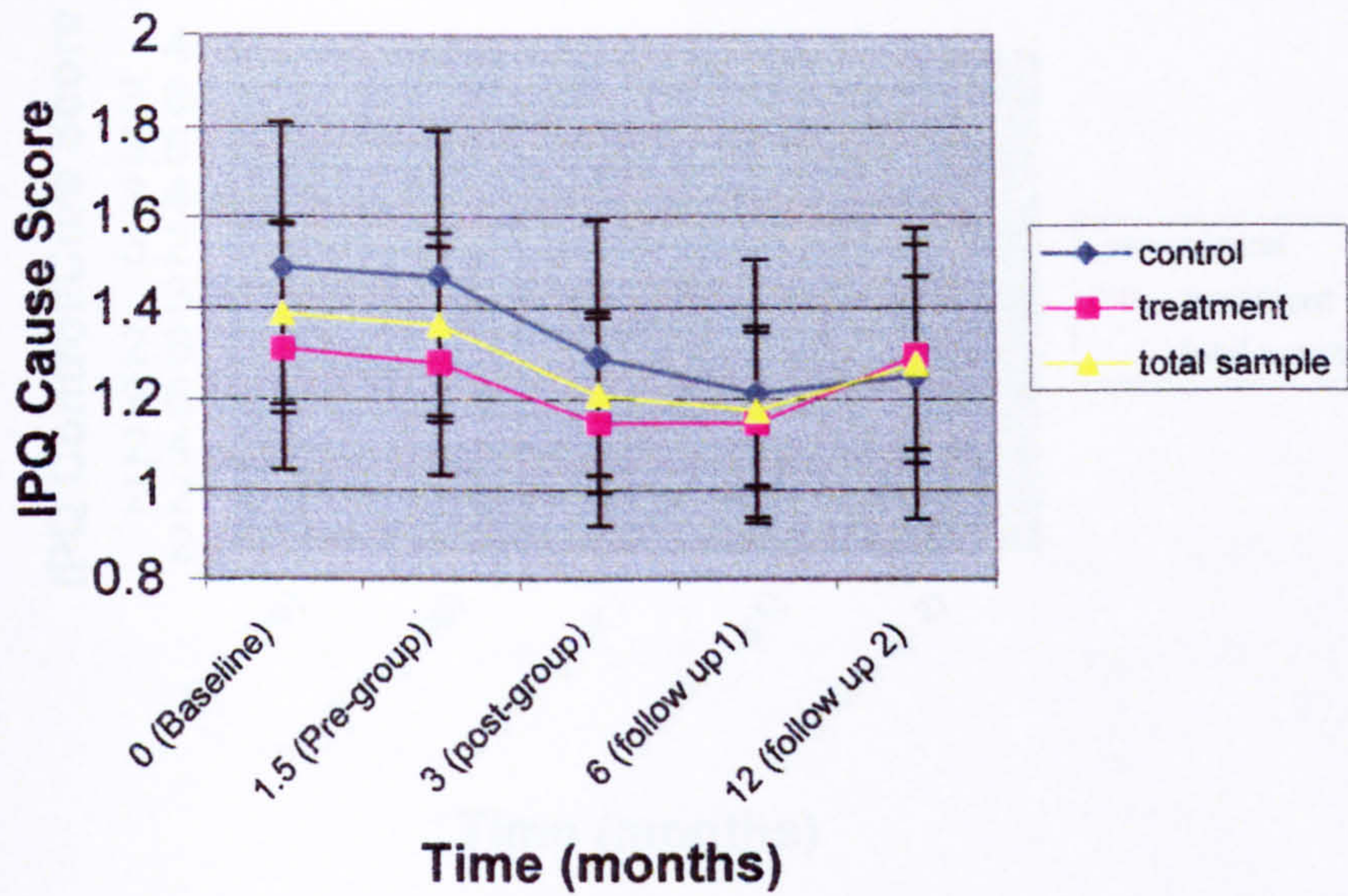
Figure 7.25: Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Consequences scores between time 1 & 5



7.3(e) Cause

For IPQ Cause there were no significant results on the main ANOVA and ANCOVA. For the planned contrasts there was a significant within participants effect for time 1 vs 4 ($F = 4.510$, $df = 1$, $p = 0.036^*$). Figure 7.26 shows that overall the control group identify slightly more factors that they believe played a role in causing their RA (make slightly more causal attributions). Both groups show a decrease in the number of causal attributions between time 1 and 4 and the treatment group returns to baseline at time 5.

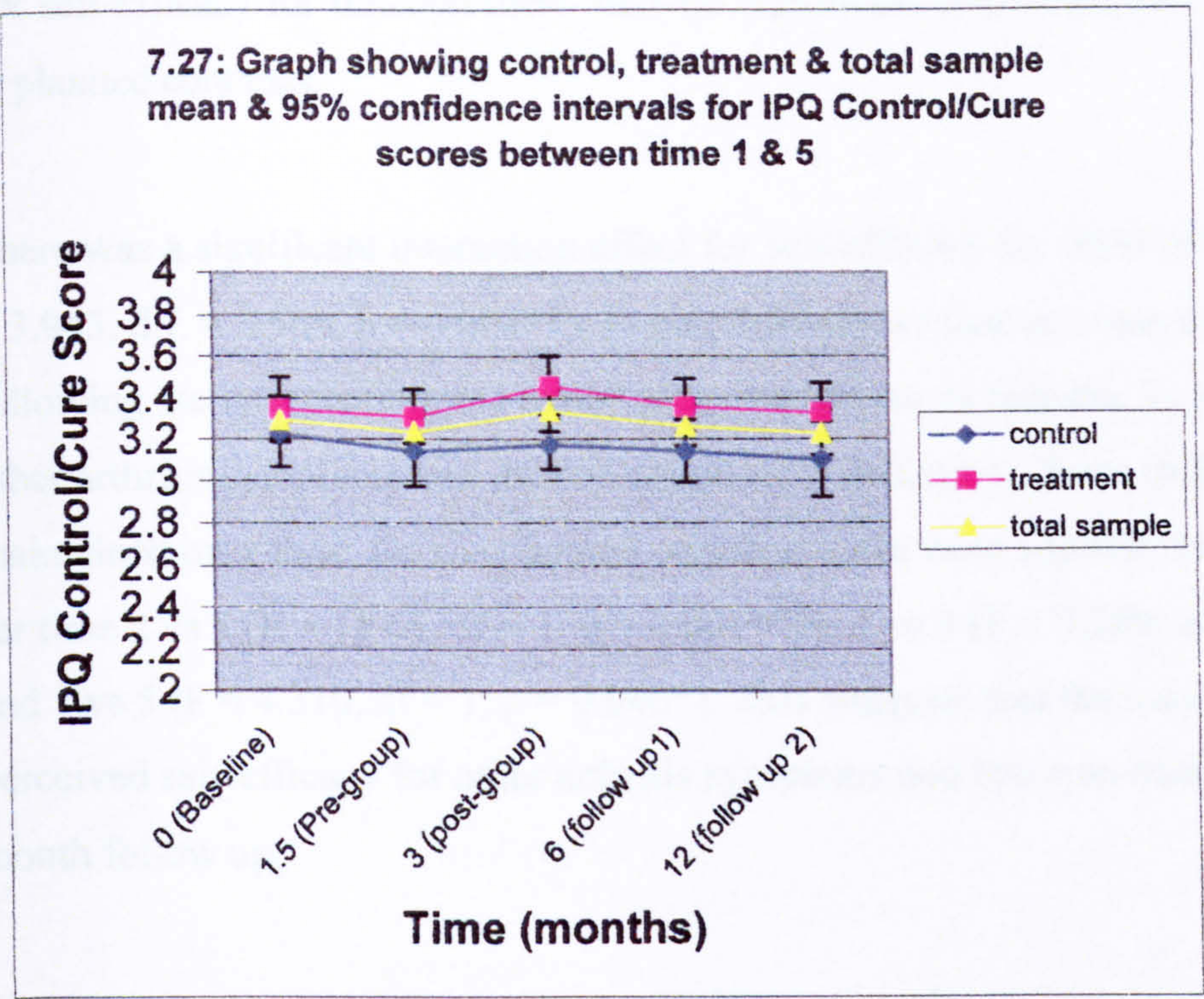
Figure 7.26: Graph showing control, treatment & total sample mean & 95% confidence intervals for IPQ Cause scores between time 1 & 5



7.3(f) Control/cure

There was a significant between participants main effect for IPQ Control/Cure ($F = 5.457$, $df = 1$, $p = 0.021^*$). This was not significant on the ANCOVA ($F = 3.625$, $df = 1$ $p = 0.060$). On the planned contrasts there was a significant within participants effect for time 2 vs 3 ($F = 3.81$, $df = 1$, $p = 0.050^*$).

Figure 7.27 shows that the treatment group had higher perceived control/cure beliefs throughout the study. The treatment group shows an increase in perceived control/ cure between time 2 and 3, following the intervention, but returns to baseline by time 4. The control group shows little change.



7.3(g) Self-efficacy

For self-efficacy for pain there was a significant within participants effect ($F = 3.372$, $df = 3.733$, $p = 0.012^*$). The within participants effect was not significant on the ANCOVA ($F = 1.377$, $df = 3.728$, $p = 0.243$). There was a significant interaction effect for self-efficacy for pain ($F = 4.471$, $d.f. = 3.733$, $p = 0.002^{**}$). On Figure 7.28 the treatment group shows a significant increase in self-efficacy for pain after the intervention, at time 3, which is maintained over time. The control group shows little change over time.

On the planned contrasts there were significant within participants effects for time 2 vs 3 ($F = 8.870$, $df = 1$, $p = 0.004^{**}$) and 1 vs 3 ($F = 6.585$, $df = 1$, $p = 0.012^*$). There were significant interaction effects for time 2 vs 3 ($F = 9.532$, $df = 1$, $p = 0.003^{**}$), 1 vs 3 ($F = 10.232$, $df = 1$, $p = 0.002^{**}$) and 1 vs 4 ($F = 6.097$, $df = 1$, $p = 0.015^*$). This suggests that the intervention increased perceived self-efficacy for pain and this was maintained during the 9-month follow up period.

For self-efficacy for function there were no significant results for the ANOVA, ANCOVA or planned contrasts.

There was a significant interaction effect for self-efficacy for other arthritis symptoms ($F = 3.943$, d.f. = 3.509, $p = 0.004^{**}$). Figure 7.30 shows that between time 2 and 3, following the intervention, the treatment group shows an increase in self-efficacy for other arthritis symptoms and the control group a reduction. These differences are maintained over time. On the planned contrasts there were significant interaction effects for time 2 vs 3 ($F = 11.65$, $df = 1$, $p = 0.001^{***}$), 1 vs 3 ($F = 7.289$, $df = 1$, $p = 0.008^{**}$) and 1 vs 5 ($F = 4.310$, $df = 1$, $p = 0.040^{*}$). This suggests that the intervention increased perceived self-efficacy for other arthritis symptoms and this was maintained at the 9 month follow up.

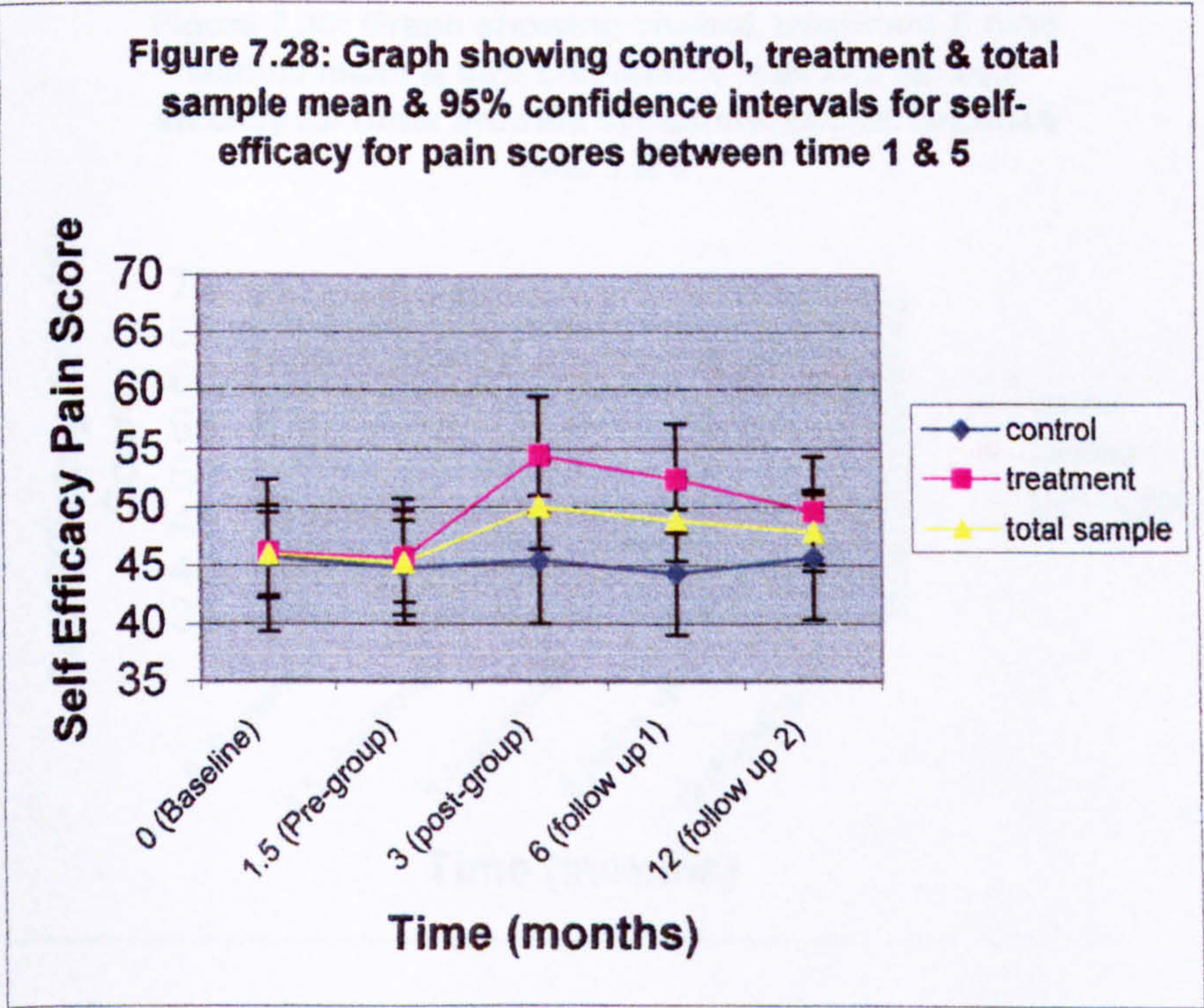


Figure 7.29: Graph showing control, treatment & total sample mean & 95% confidence interval for self-efficacy for function scores between time 1 & 5

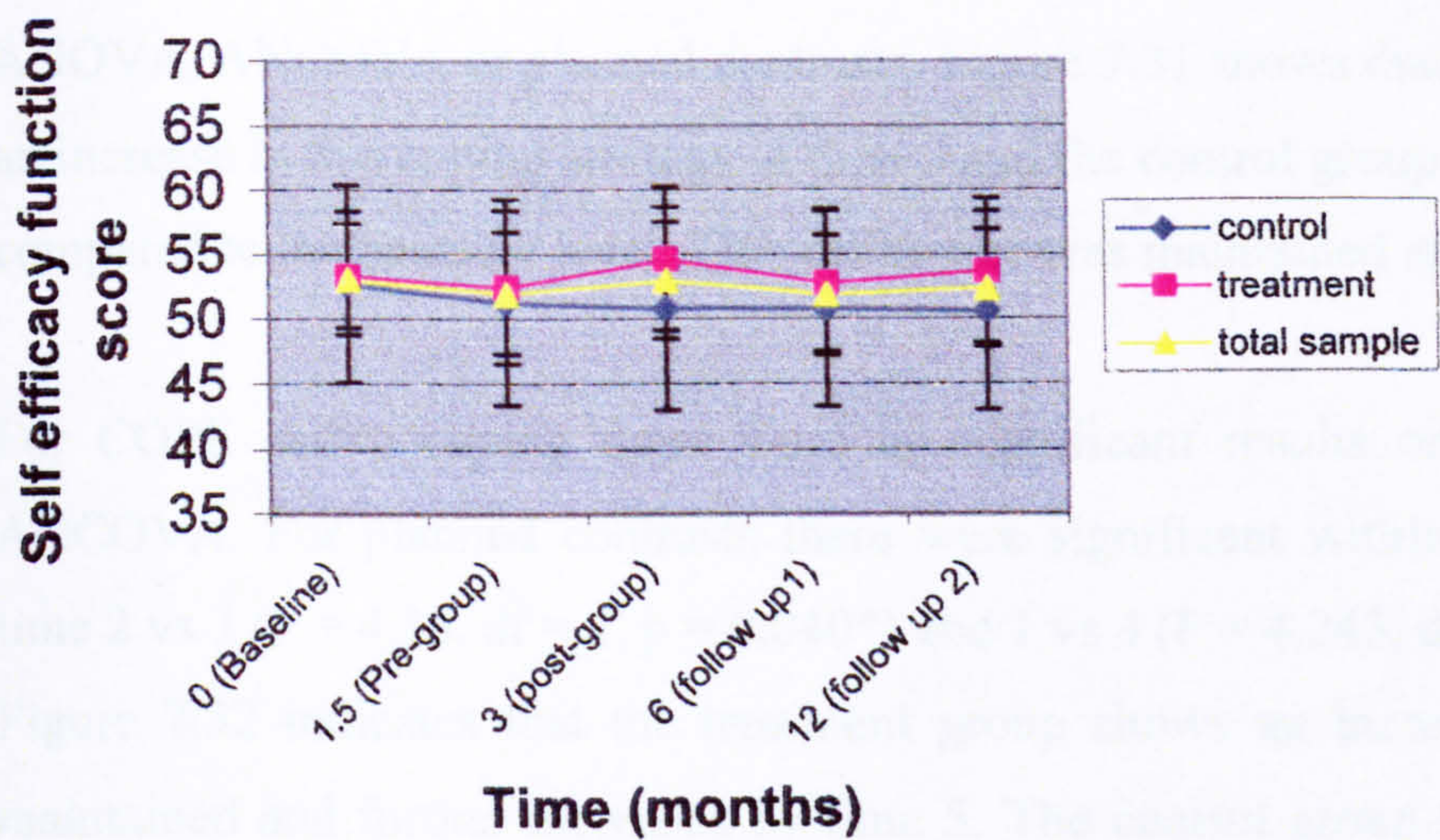
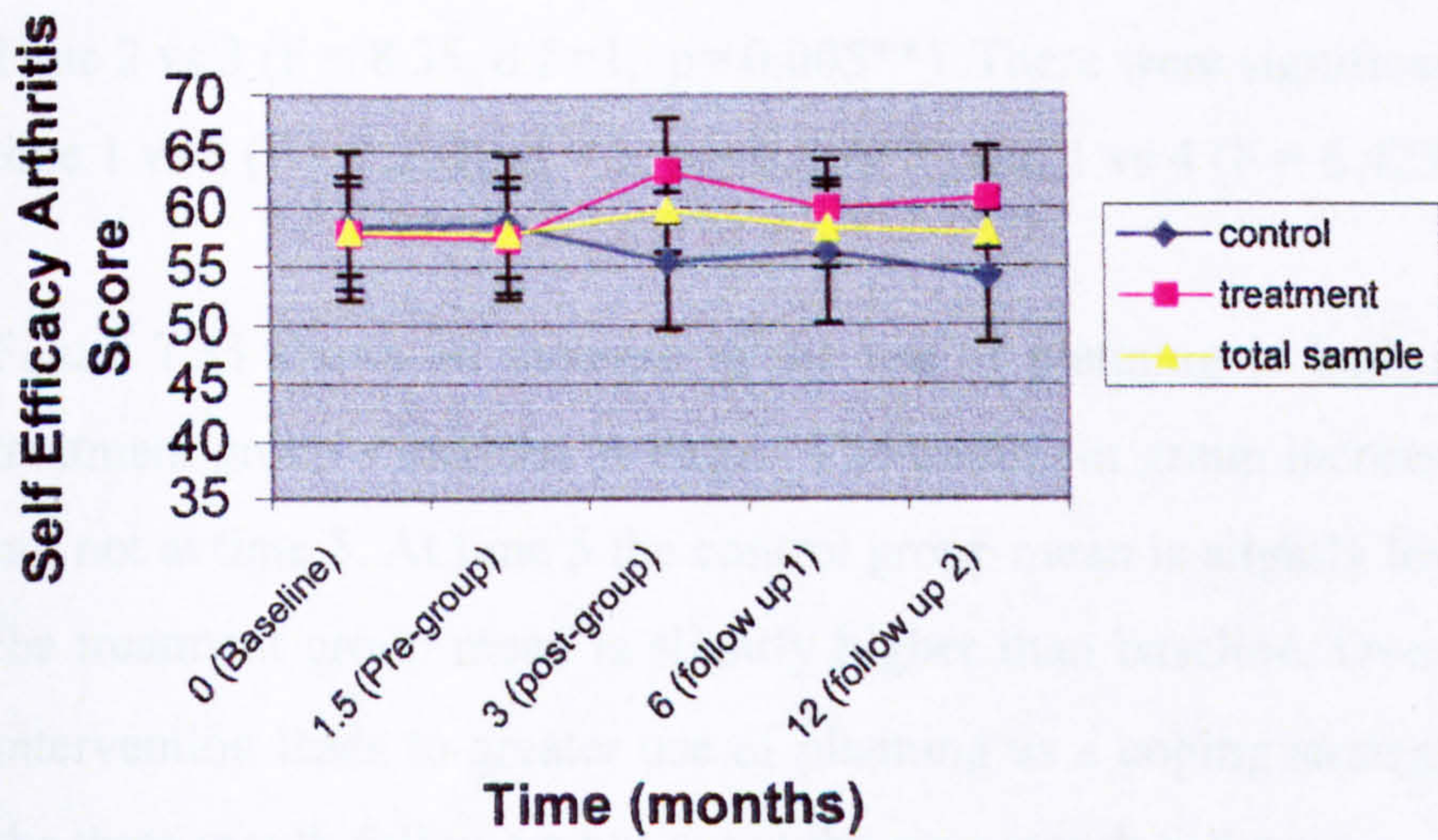


Figure 7.30: Graph showing control, treatment & total sample mean & 95% confidence intervals for self-efficacy for other arthritis symptoms scores between time 1 & 5



7.3(h) *Coping strategies*

For COPE positive interpretation/growth there were no significant results on the main ANOVA, ANCOVA or planned contrasts. Figure 7.31 shows that the treatment group has an increase in this coping strategy at time 3 and the control group shows a decrease compared to the baseline level. This difference was maintained at the end of the study.

For COPE active coping there were no significant results on the main ANOVA or ANCOVA. For planned contrasts there were significant within participants effects for time 2 vs 3 ($F = 4.38$, $df = 1$, $p = 0.040^*$) and 1 vs 4 ($F = 4.243$, $df = 1$, $p = 0.042^*$). Figure 7.32 indicates that the treatment group shows an increase at time 2, which is maintained and further increases by time 5. The control group shows little change over time.

For COPE planning there was a significant interaction effect ($F = 3.340$, $df = 3.542$, $p = 0.014^*$). The planned contrasts show a significant within participants main effect for Time 2 vs 3 ($F = 8.35$, $d.f.=1$, $p= 0.005^{**}$). There were significant interaction effects for time 1 vs 3 ($F = 7.252$, $df = 1$, $p = 0.008^{**}$) and 1 vs 4 ($F = 6.423$, $df = 1$, $p = 0.013^*$).

Figure 7.33 shows an increase in the use of planning in both groups at time 3 but the treatment group's increase is larger. The treatment group increase is maintained at time 4 but not at time 5. At time 5 the control group mean is slightly lower than the baseline and the treatment group mean is slightly higher than baseline. Overall this suggests that the intervention leads to greater use of planning as a coping strategy which is maintained at the three month follow up but not at the nine month follow up.

For COPE seeking emotional social support there was a significant between participants main effect ($F = 8.574$, $df = 1$, $p = 0.004^{**}$). There was a significant within participant main effect ($F = 2.993$, $df = 3.321$, $p = 0.026^*$) but this was not significant on the ANCOVA ($F = 1.290$, $df = 3.332$, $p = 0.276$). On the planned contrasts there were

significant within participants effects for time 1 vs 2 ($F = 7.990$, $df = 1$, $p = 0.006^{**}$), 2 vs 3 ($F = 4.28$, $df = 1$, $p = 0.04^{*}$) and 1 vs 5 ($F = 5.172$, $df = 1$, $p = 0.025^{*}$).

Figure 7.34 shows that the treatment group uses this strategy more. During the study both groups show a slight reduction in the use of the strategy although there is a slight increase at time 2 for both groups and at time 3 for the treatment group.

For COPE seeking instrumental social support there was a significant between participants main effect ($F = 8.066$, $df = 1$, $p = 0.005^{**}$). There was a significant within participants main effect ($F = 3.844$, $df = 3.542$, $p = .006^{**}$). On the planned contrasts there were significant within participants effects for time 1 vs 2 ($F = 5.07$, $df = 1$, $p = 0.030^{*}$), 1 vs 4 ($F = 4.243$, $df = 1$, $p = 0.042^{*}$) and 1 vs 5 ($F = 12.503$, $df = 1$, $p = 0.001^{***}$).

Figure 7.35 shows that overall the treatment group uses this strategy more. Both groups use the strategy less over time although the treatment group shows a small increase at time 3.

For COPE suppression of competing activities there was a significant between participants main effect ($F = 7.239$, $df = 1$, $p = 0.008^{**}$). For the planned contrast there were significant interaction effects for time 3 vs 4 ($F = 5.690$, $df = 1$, $p = 0.020^{*}$), 4 vs 5 ($F = 5.910$, $df = 1$, $p = 0.020^{*}$) and 1 vs 4 ($F = 4.997$, $df = 1$, $p = 0.027^{*}$). Figure 7.36 shows that the treatment group uses this strategy more. Both groups show little change between time 1 and 5, but the control group shows a temporary increase at time 3 and the treatment group shows a temporary increase at time 4.

For COPE acceptance there were no significant results on the ANOVA or ANCOVA. For the planned contrasts there were significant within participants effects for time 1 vs 4 ($F = 4.805$, $df = 1$, $p = 0.030^{*}$) and 1 vs 5 ($F = 3.970$, $df = 1$, $p = 0.049^{*}$).

Figure 7.37 shows that both groups use this strategy less over time with the treatment group decreasing their use more at time 4 and 5.

For COPE mental disengagement there were no significant results on the ANOVA or ANCOVA. For the planned contrasts there was a significant interaction effect for time 4 vs 5 ($F = 4.160$, $df = 1$, $p = 0.040^*$). Figure 7.38 shows that both groups show little change between time 1 and 5. The control group do decrease and then increase their use slightly during the study. The treatment group increases their use at time 3 and 4 but return to nearly baseline levels at time 5.

For COPE focusing/venting emotions there was a significant within participants main effect ($F = 6.299$, $df = 3.112$, $p = <0.001^{***}$) but this was not significant on the ANCOVA ($F = 1.202$, $df = 3.110$, $p = 0.309$). For the planned contrasts there were significant within participants effects for time 2 vs 3 ($F = 3.860$, $df = 1$, $p = 0.050^*$), 4 vs 5 ($F = 7.960$, $df = 1$, $p = 0.006^{**}$), 1 vs 3 ($F = 5.493$, $df = 1$, $p = 0.021^*$) and 1 vs 5 ($F = 14.235$, $df = 1$, $p = <0.001^{***}$). Figure 7.39 shows that both groups, but especially the control, reduce their use of the strategy over the study. Most of the reduction occurs at time 3 and 5.

For COPE behavioural disengagement there were no significant results for the ANOVA, ANCOVA or planned contrasts. Figure 7.40 shows there is little change over time in the use of this strategy.

For COPE denial there were no significant results for the ANOVA or ANCOVA. For the planned contrasts there was a significant interaction effect for time 1 vs 5 ($F = 6.461$, $df = 1$, $p = 0.012^*$). Figure 7.41 shows that the control group uses the strategy less over time and the treatment group reports increased use at time 3 and 5.

For COPE restraint coping there was a significant between participants main effect ($F = 7.879$, $df = 1$, $p = 0.006^{**}$). For the planned contrasts there was a significant within participants effect for time 3 vs 4 ($F = 4.280$, $df = 1$, $p = 0.040^*$).

Figure 7.42 shows that the treatment group uses this strategy more. The control group shows a reduction in use over time with a slight decrease at time 3 and increase at 4. The treatment group shows little change over time but has a slight decrease at time 3 and 5.

For COPE humour there was a significant interaction effect ($F = 2.667$, $df = 3.385$, $p = 0.041^*$). This was not significant on the ANCOVA ($F = 2.273$, $df = 3.386$, $p = 0.072$). The planned contrasts show significant interaction effects between time 1 and 2 ($F = 4.89$, $df = 1$, $p = 0.030^*$), time 1 and 4 ($F = 4.732$, $df = 1$, $p = 0.032^*$) and time 1 and 5 ($F = 6.878$, $df = 1$, $p = 0.010^{**}$).

Figure 7.43 shows that the control group reports using humour less over time and the treatment group reports using humour more over time. The largest difference between the groups occurs between time 1 and 2. The treatment group reports increased use of humor and the control group decreased use of humour between time 1-2, 3-4 and 4-5.

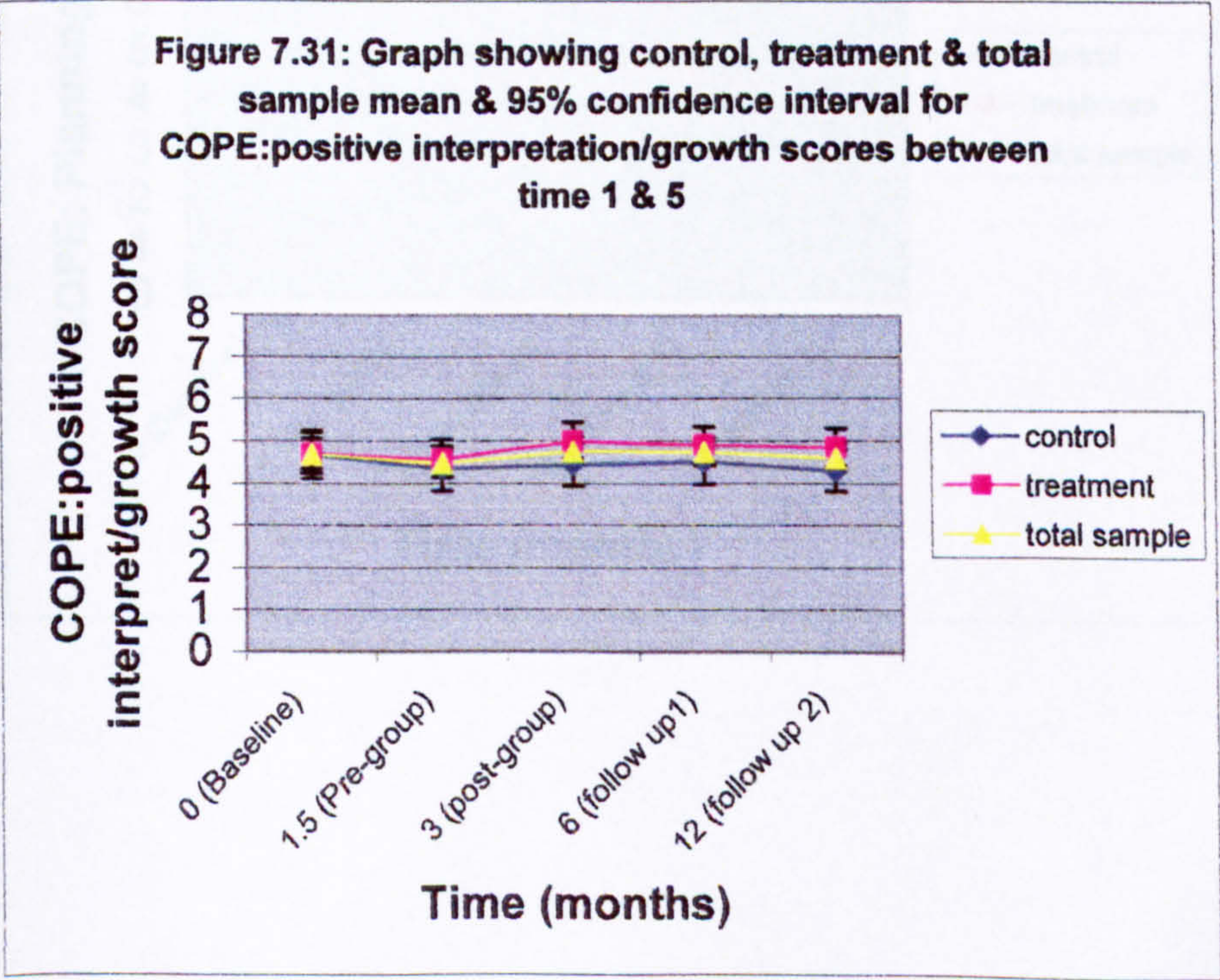


Figure 7.32: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE:Active coping scores between time 1 & 5

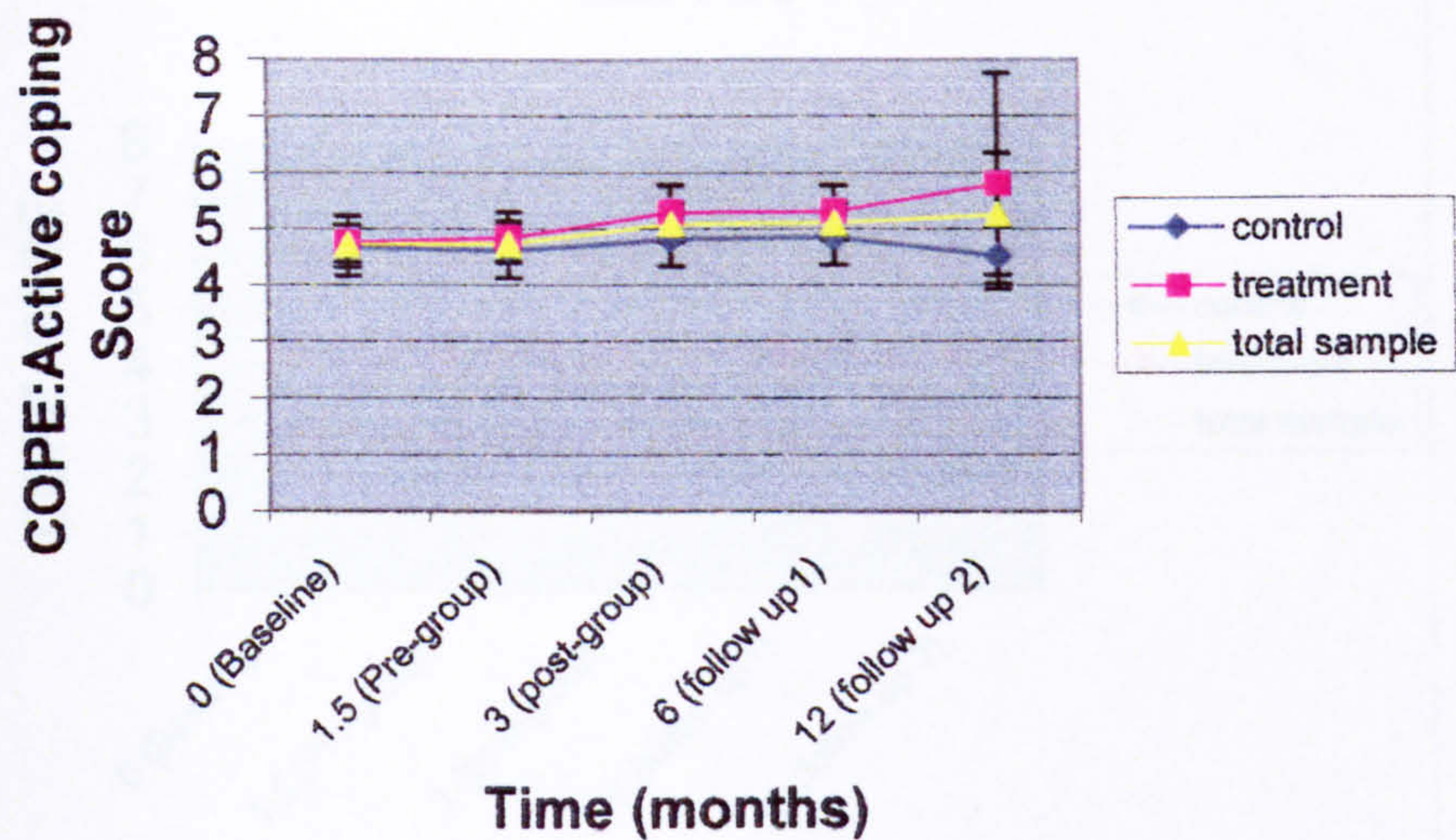


Figure 7.33: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Planning scores between time 1 & 5

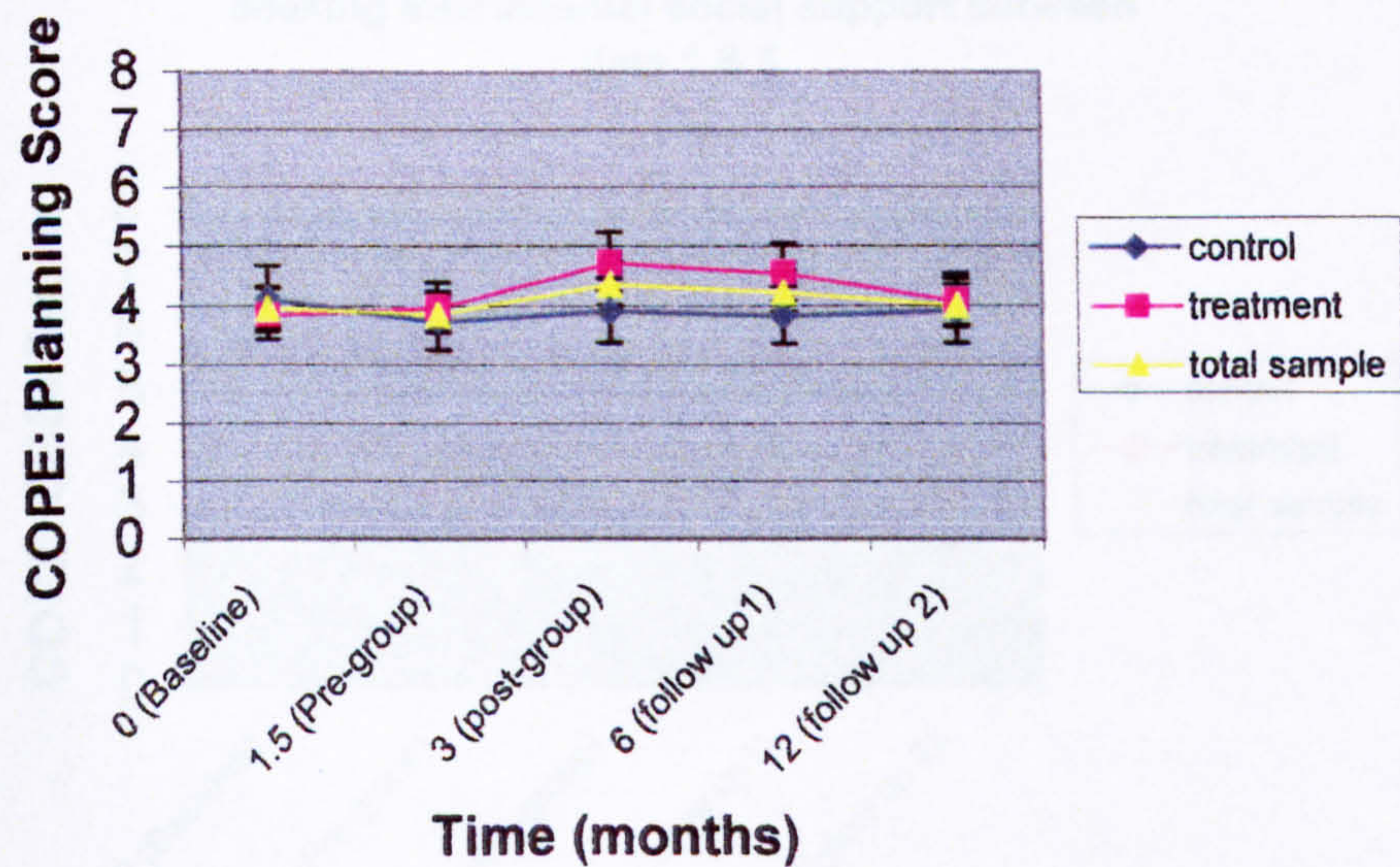


Figure 7.34: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Seeking emotional social support scores between time 1 & 5

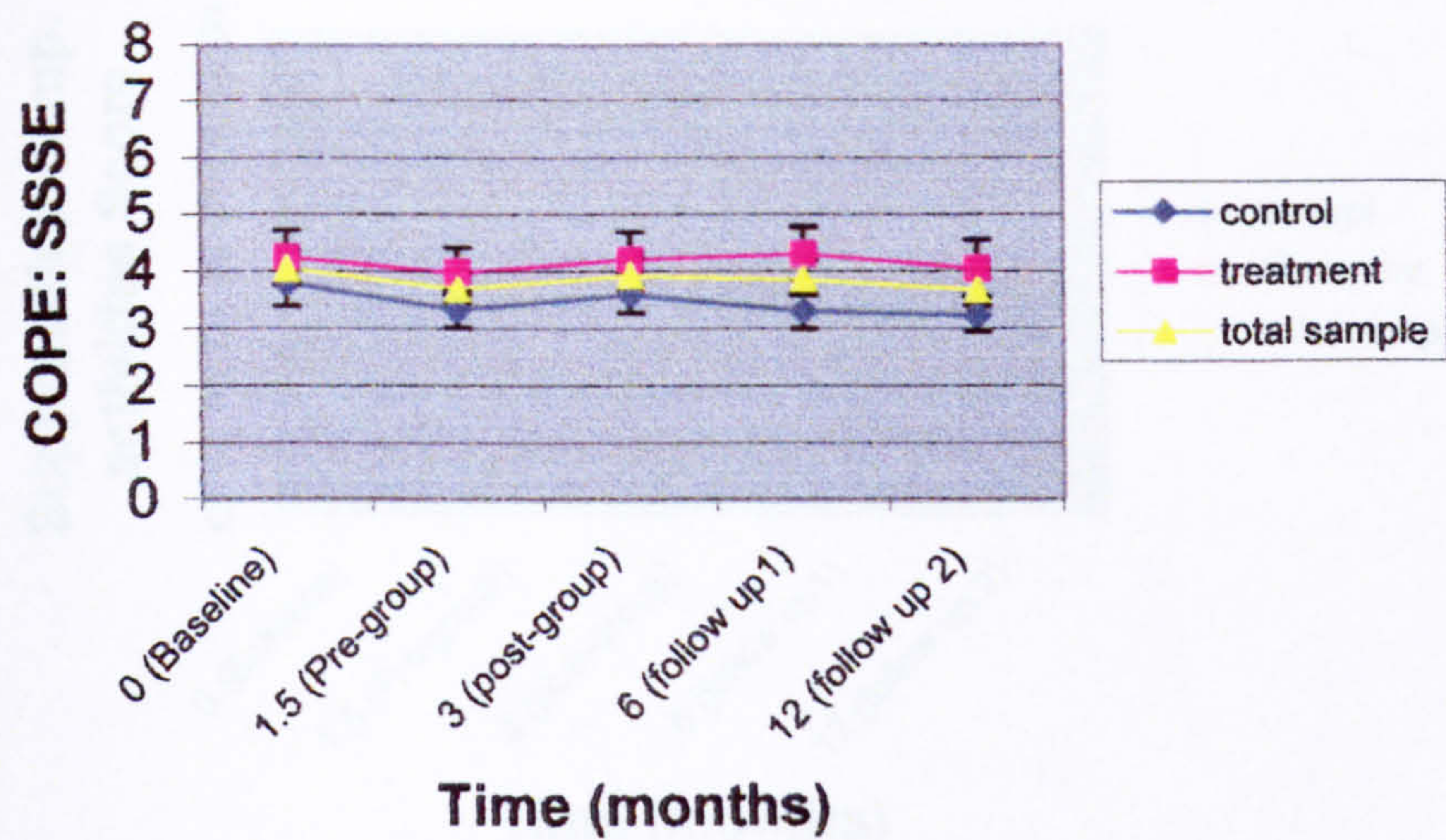


Figure 7.35: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Seeking instrumental social support between time 1 & 5

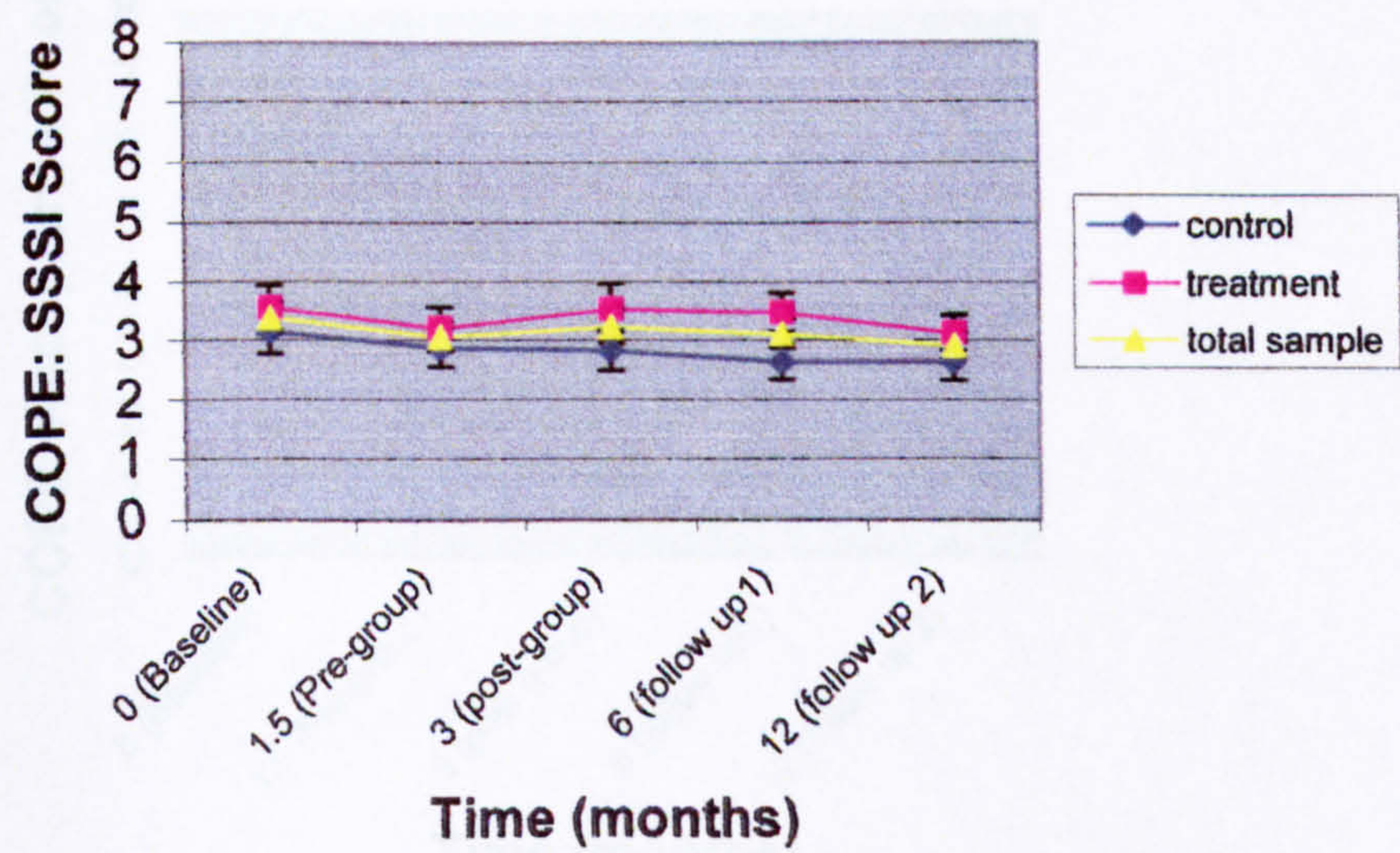


Figure 7.36: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Suppression of competing activities scores between time 1 & 5

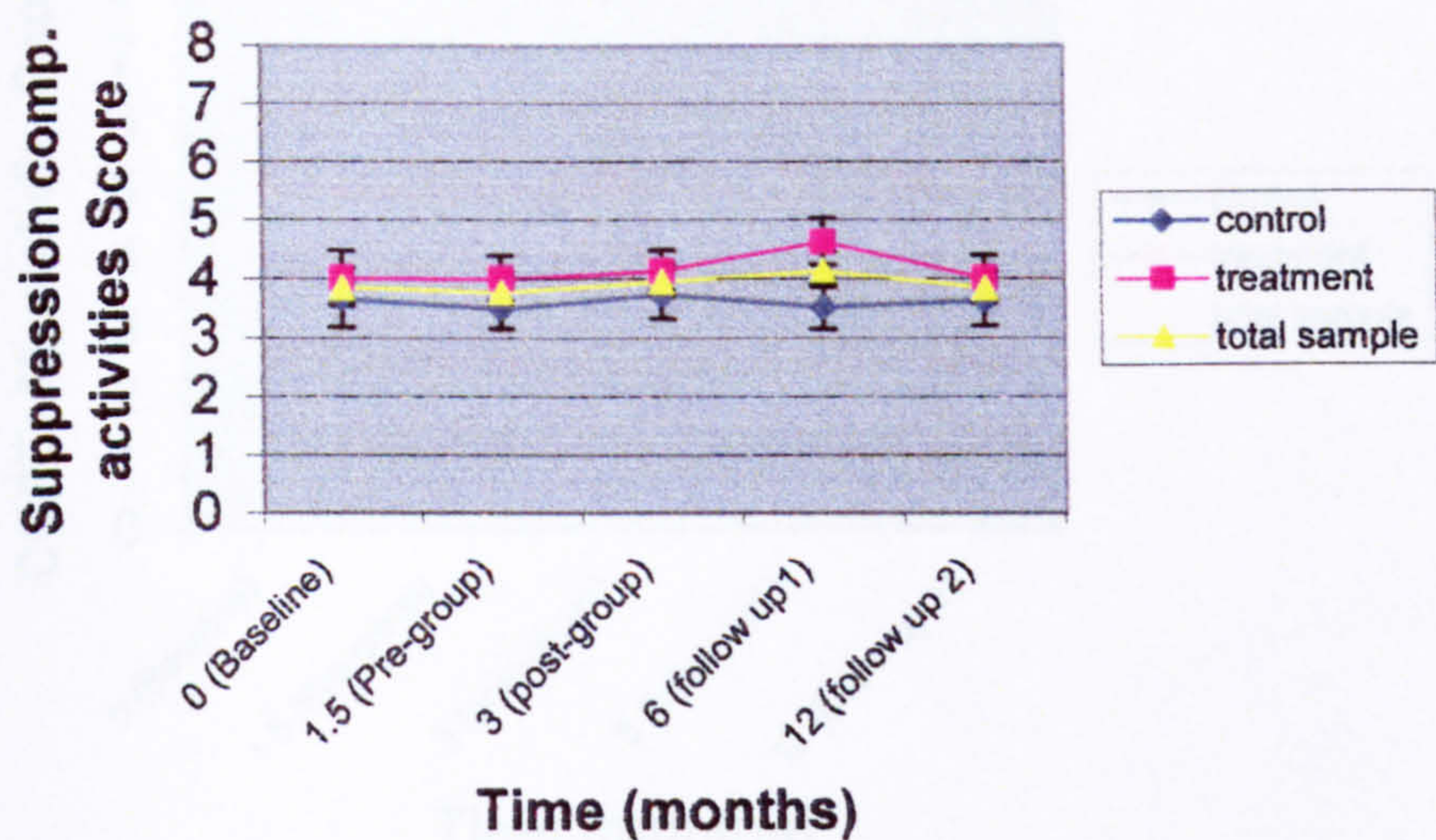


Figure 7.37: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Acceptance scores between time 1 & 5

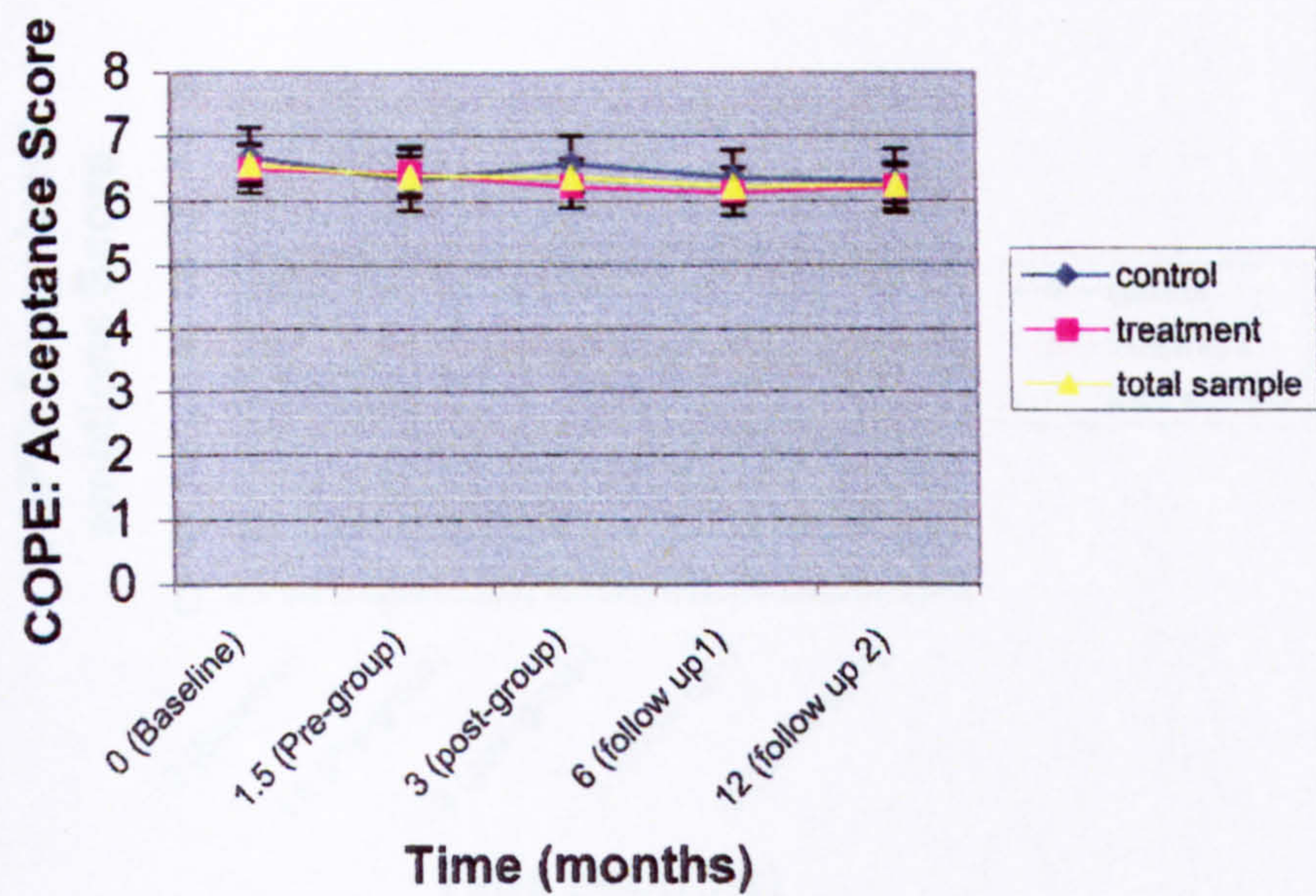


Figure 7.38: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Mental disengagement scores between time 1 & 5

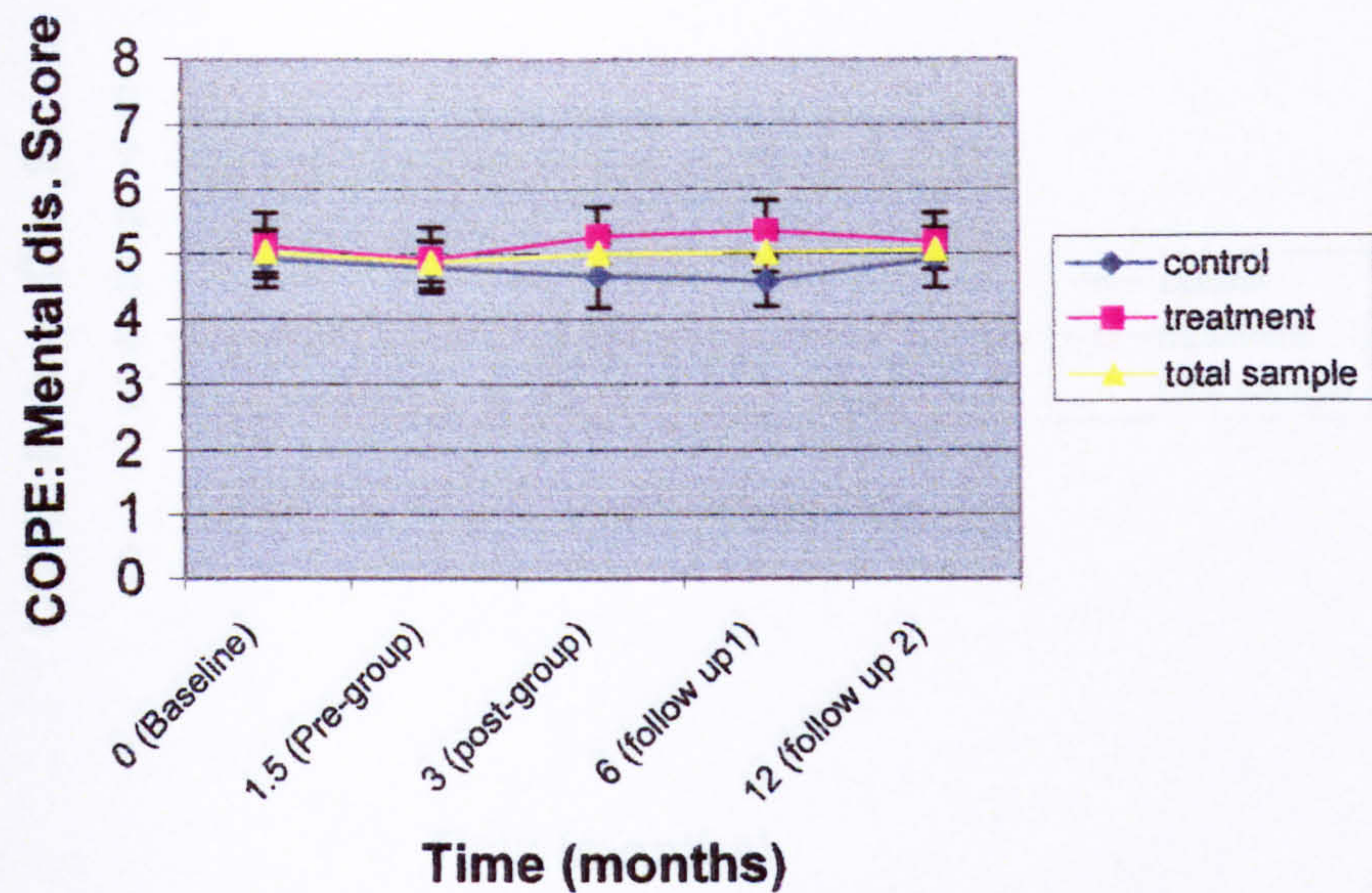


Figure 7.39: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Focus/vent emotions scores between time 1 & 5

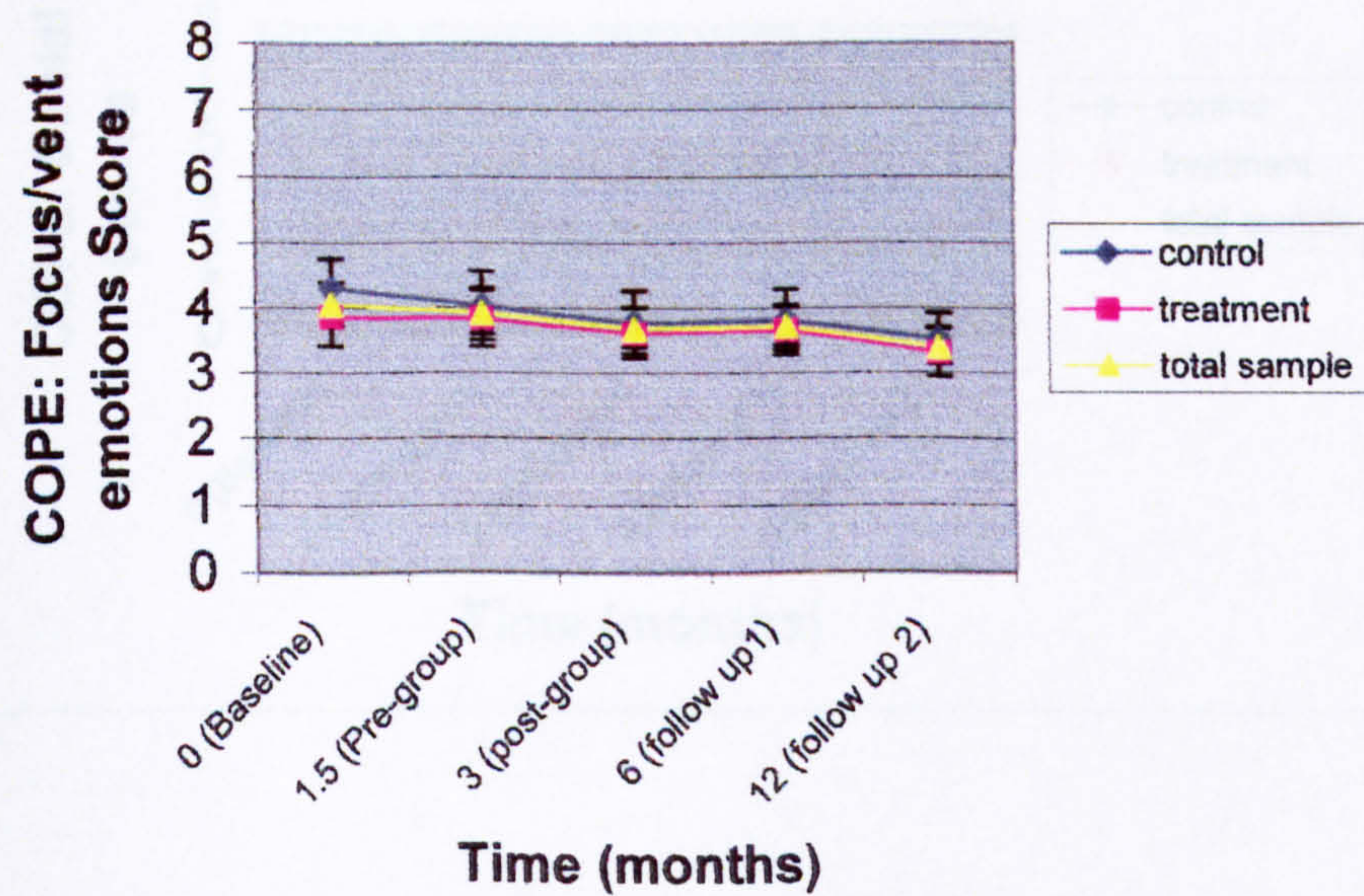


Figure 7.40: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Behavioural disengagement scores between time 1 & 5

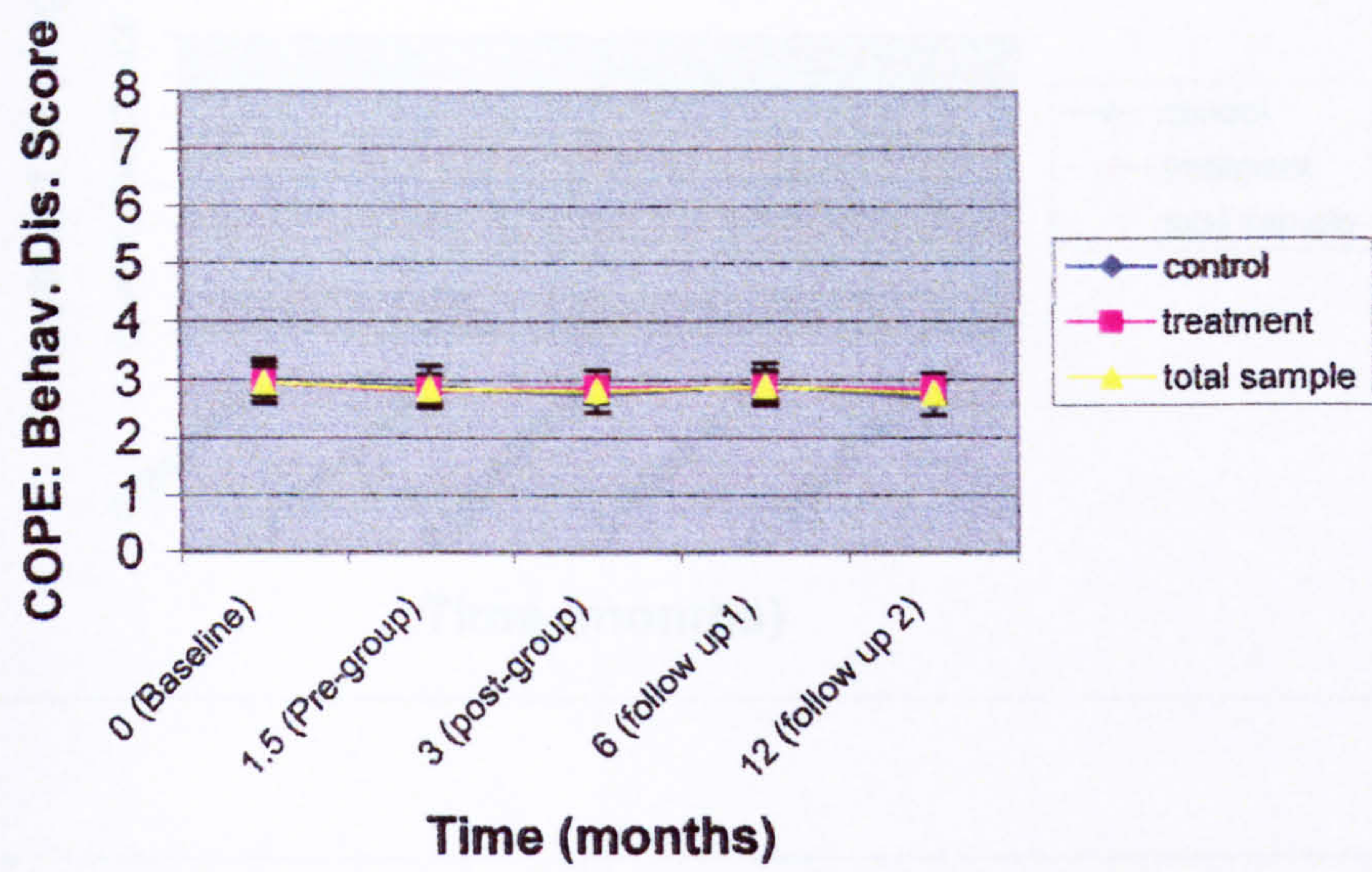


Figure 7.41: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Denial scores between time 1 & 5

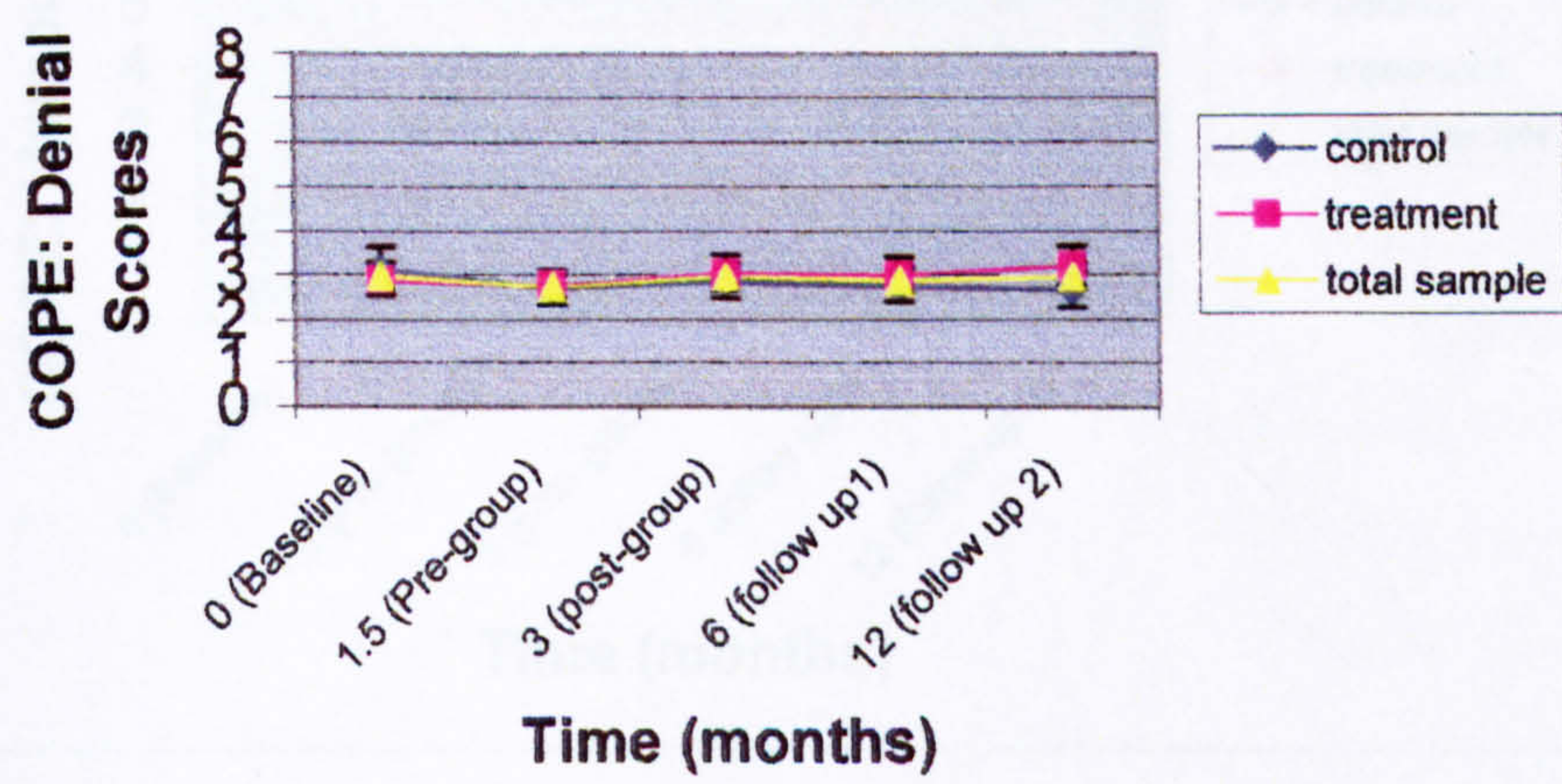


Figure 7.42: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Restraint coping between time 1 & 5

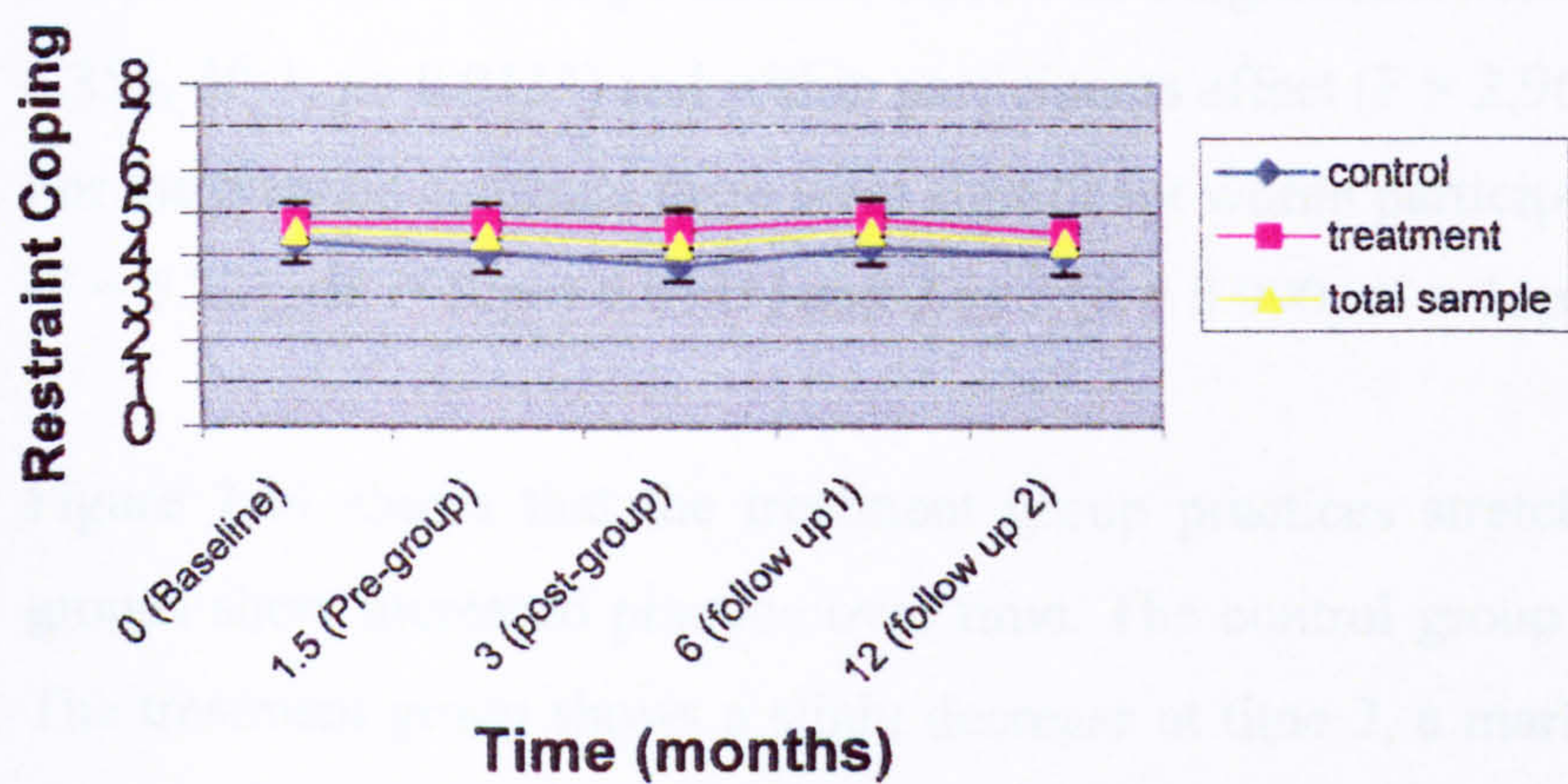
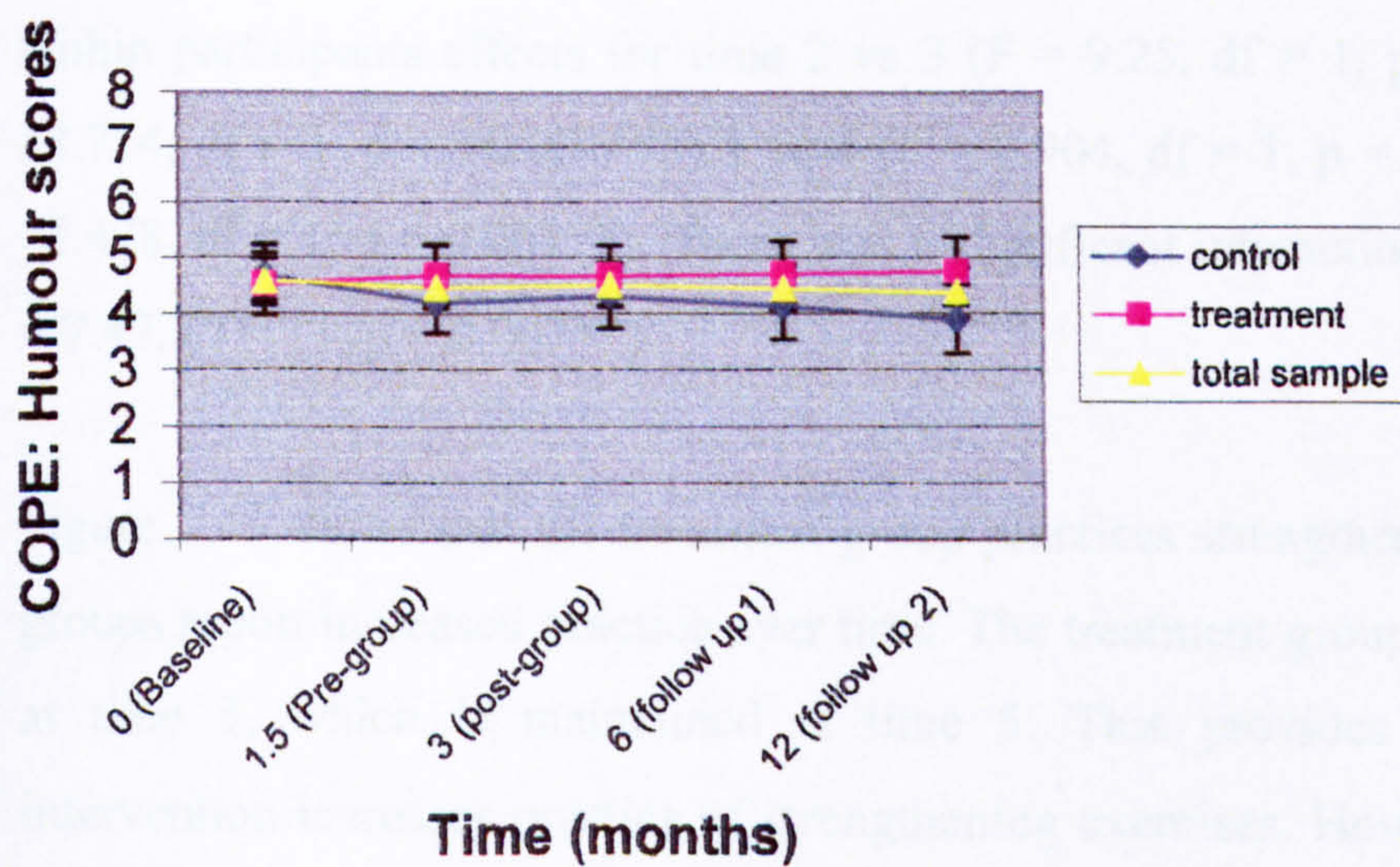


Figure 7.43: Graph showing control, treatment & total sample mean & 95% confidence intervals for COPE: Humour scores between time 1 & 5



7.3(i) *Self management behaviours*

For practice of stretching exercises there was a significant between participants effect ($F=6.354$, $df=1$, $p=0.013^*$) and within participants effect ($F=2.967$, $df=3.576$, $p=0.024^*$). For the planned contrasts there were significant within participants effects for time 1 vs 3 ($F=5.721$, $df=1$, $p=0.018^*$) and 2 vs 3 ($F=8.990$, $df=1$, $p=0.003^{**}$).

Figure 7.44 shows that the treatment group practices stretching exercises more. Both groups show increased practice over time. The control group highest score is at time 3. The treatment group shows a slight decrease at time 2, a marked increase at time 3, and slight reductions at time 4 and 5.

For practice of strengthening exercises There was a significant between participants effect ($F=8.712$, $d.f.=1$, $p=0.004^{**}$). There was a significant within participants effect ($F=6.190$, $df=3.444$, $p<0.001^{***}$). For the planned contrasts there were significant within participants effects for time 2 vs 3 ($F=9.25$, $df=1$, $p=0.003^{**}$), 1 vs 3 ($F=18.754$, $df=1$, $p<0.001^{***}$), 1 vs 4 ($F=9.904$, $df=1$, $p=0.002^{**}$) and 1 vs 5 ($F=11.478$, $df=1$, $p=0.001^{**}$). There was a significant interaction effect for time 2 vs 3 ($F=7.47$, $df=11$, $p=0.007^{**}$).

Figure 7.45 shows that the treatment group practices strengthening exercises more. Both groups report increased practice over time. The treatment group shows a marked increase at time 3, which is maintained at time 5. This provides some evidence that the intervention increases practice of strengthening exercises. However as the control group also improves to some extent the difference between the groups is not sufficient to produce a significant interaction effect on the main ANOVA

For practice of relaxation there was a significant within participants effect ($F=6.756$, $df=3.243$, $p<0.001^{***}$). This was not significant on the ANCOVA ($F=2.077$, $df=3.208$, $p=0.099$). For planned contrasts there were significant within participants effects for time 2 vs 3 ($F=21.29$, $df=1$, $p<0.001^{***}$), 1 vs 3 ($F=12.523$, $df=1$, $p=0.001^{**}$)

and 1 vs 4 ($F = 5.170$, $df = 1$, $p = 0.025^*$). Figure 7.46 shows that the control group changed little over time, although there was a slight decrease at time 2 and increase at time 3. The treatment group shows a marked increase at time 3, which is maintained at 4 and 5.

For Practice of Massage there were no significant results for the ANOVA or ANCOVA. For the planned contrast there was a significant within participants effect for time 2 vs 3 ($F = 4.16$, $df = 1$, $p = 0.040^*$). Figure 7.47 shows a slight decrease in practice of massage in both groups over time.

There was a significant within participants main effect for the frequency of walks over half a mile each week ($F = 3.840$, $df = 3.282$, $p = 0.008^{**}$) and this is not significant on the ANCOVA ($F = 0.617$, $df = 3.340$, $p = 0.622$). For the planned contrasts there were significant within participants effects for time 1 vs 2 ($F = 6.070$, $df = 1$, $p = 0.020^*$), 1 vs 3 ($F = 4.365$, $df = 1$, $p = 0.039^*$), 1 vs 4 ($F = 8.894$, $df = 1$, $p = 0.003^{**}$) and 1 vs 5 ($F = 10.316$, $df = 1$, $p = 0.002^{**}$). Figure 7.48 shows that both groups increase the frequency of walks over half a mile during the study.

For number of minutes walked per week there were no significant results on the ANOVA or ANCOVA. For the planned contrasts there were significant within participants effects for time 1 vs 2 ($F = 5.880$, $df = 1$, $p = 0.020^*$) and 1 vs 4 ($F = 6.827$, $df = 1$, $p = 0.010^{**}$). Figure 7.49 shows that both groups increase the number of minutes walked per week over the study with the control group showing most improvement at time 2 and 4 and the treatment group improving at time 2.

There was a significant within participants main effect for number of miles walked per week ($F = 2.654$, $d.f. = 3.081$, $p = 0.047^*$) and this is not significant on the ANCOVA ($F = 0.640$, $df = 3.017$, $p = 0.590$). For planned contrasts there were significant within participants effects for time 1 vs 3 ($F = 6.836$, $df = 1$, $p = 0.010^{**}$), 1 vs 4 ($F = 9.475$, $df = 1$, $p = 0.003^{**}$) and 1 vs 5 ($F = 7.041$, $df = 1$, $p = 0.009^{**}$). Figure 7.50 shows that both

groups increase the number of miles they walk per week during the study with most improvement happening in the first three months.

For frequency of cycling there were no significant results on the ANOVA, ANCOVA or planned contrasts. Figure 7.51 shows that the control group changes little over time but the treatment group shows an improvement up to time 4, whereas at time 5 it shows a decline.

For number of minutes cycling there were no significant results on the ANOVA. There was a significant within participants effect on the ANCOVA ($F = 3.815$, $df = 2.420$, $p = 0.017^*$). There were no significant results on the planned contrasts. Figure 7.52 shows a slight increase over time for both groups.

On the Frequency of Swimming there were no significant results on the ANOVA, ANCOVA or planned contrasts. Figure 7.53 shows the control group changes little over time. The treatment group shows a slight increase over time.

For the number of minutes swimming per week there was a significant within participants effect ($F=3.006$, $d.f.= 3.471$, $p= 0.024^*$). This was not significant on the ANCOVA ($F = 1.889$, $df = 3.379$, $p = 0.123$). For the planned contrasts there were significant within participants effects for time 2 vs 3 ($F= 7.760$, $df = 1$, $p = 0.006^{**}$), 1 vs 4 ($F = 0.591$, $df = 1$, $p = <0.001^{***}$) and 1 vs 5 ($F = 5.574$, $df = 1$, $p = 0.020^*$).

Figure 7.53 shows that both groups improve over time. For the control group the pattern is erratic with a decrease at time 2, increase at time 3, decrease at time 4 and increase at time 5. The treatment group shows most improvement following the intervention and the improvement is maintained at time 4 and 5.

Figure 7.44: Graph showing control, treatment & total sample mean & 95% confidence intervals for Stretching exercises scores between time 1 & 5

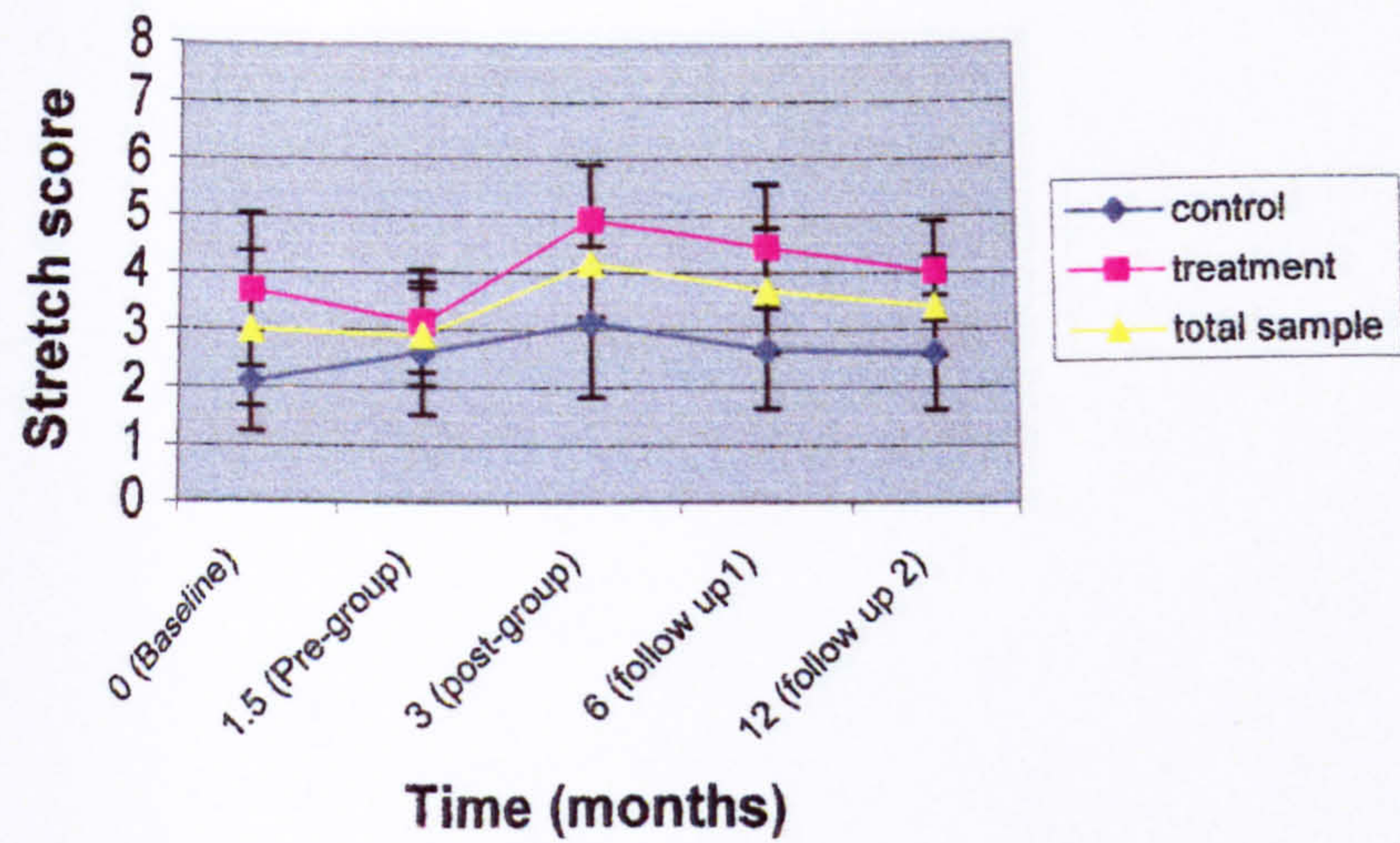


Figure 7.45: Graph showing control, treatment & total sample mean & 95% confidence intervals for Strengthening exercises scores between time 1 & 5

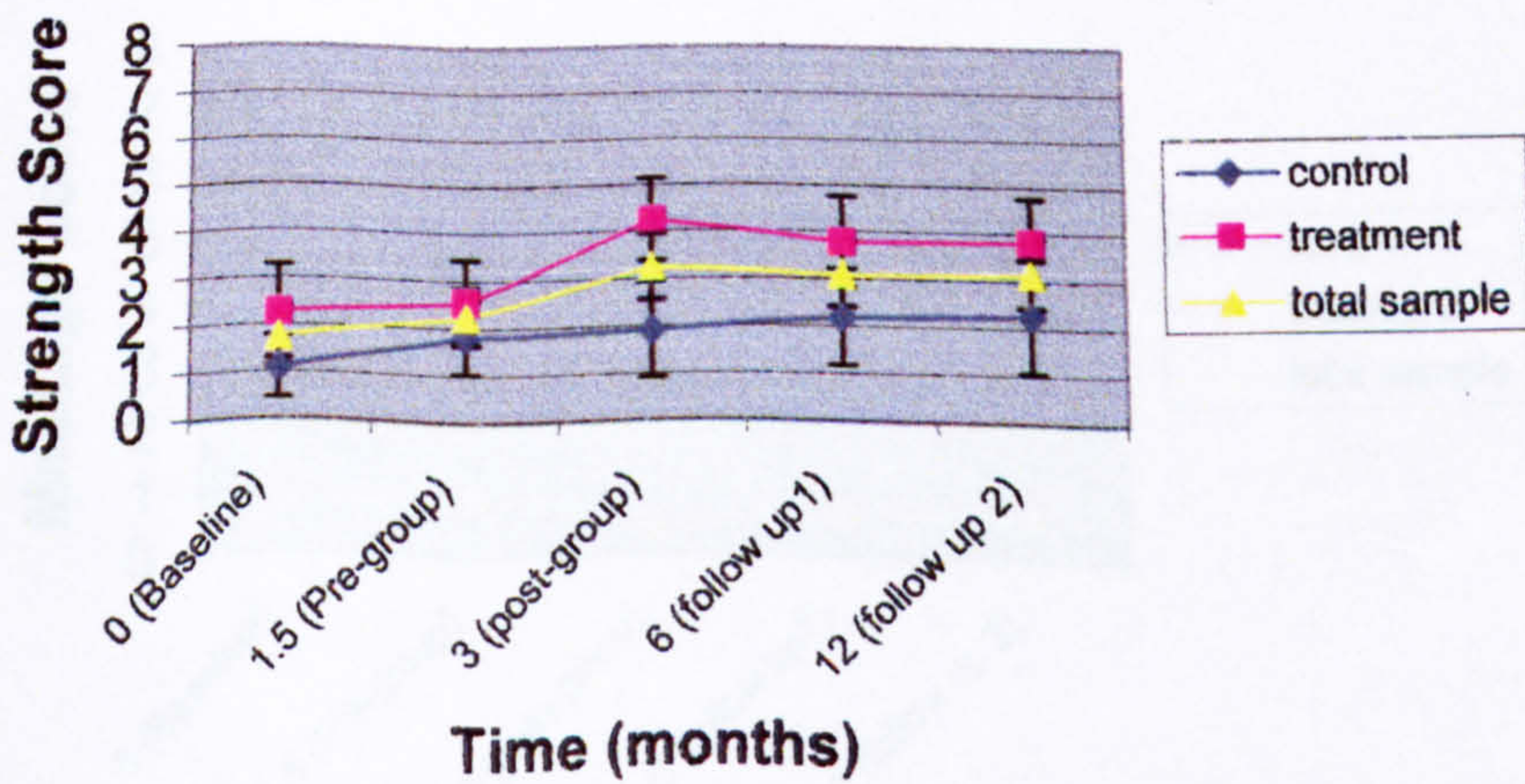


Figure 7.46: Graph showing control, treatment & total sample mean & 95% confidence interval for Relaxation scores between time 1 & 5

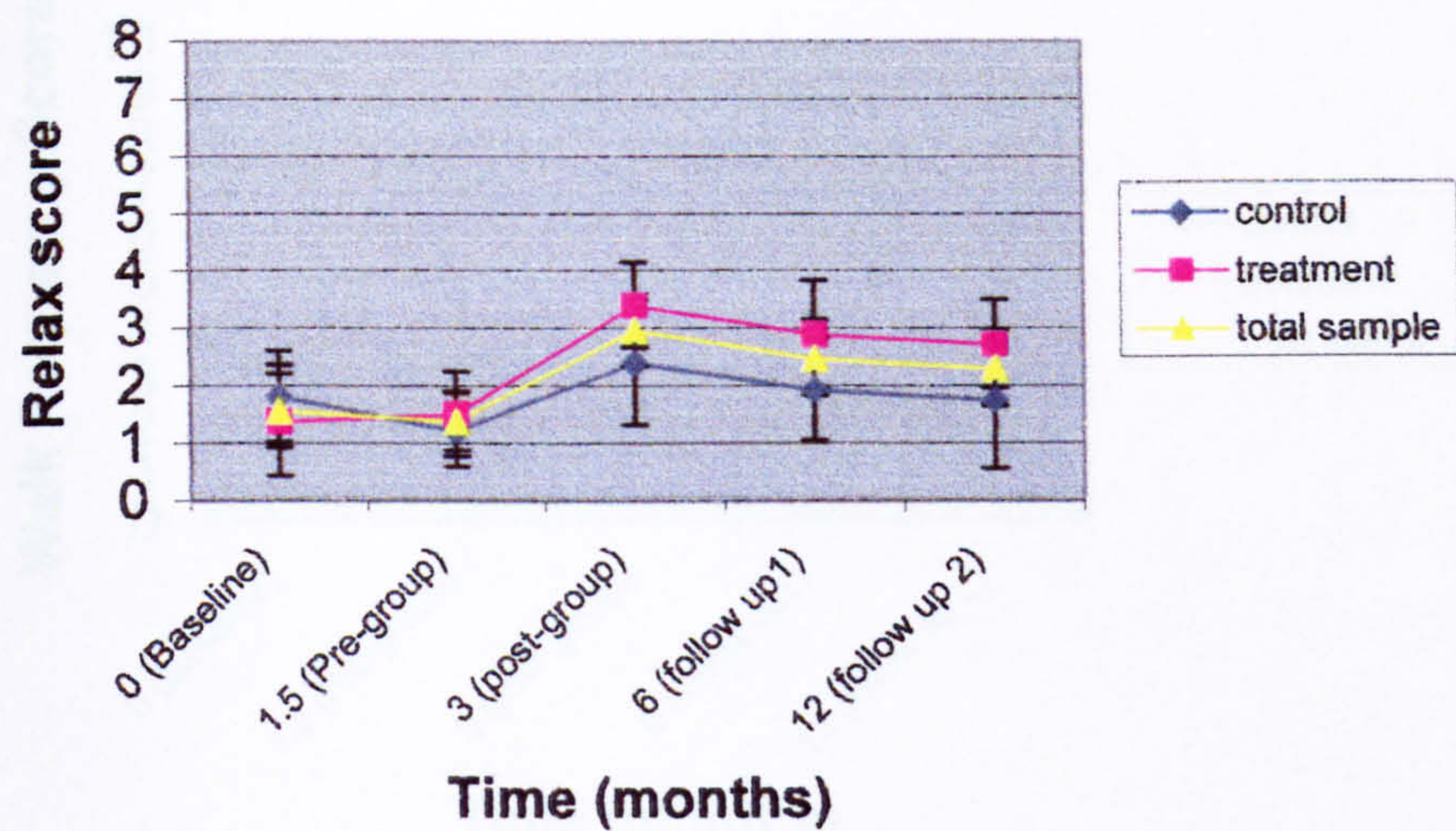


Figure 7.47: Graph showing control, treatment & total sample mean & 95% confidence intervals for Massage scores between time 1 & 5

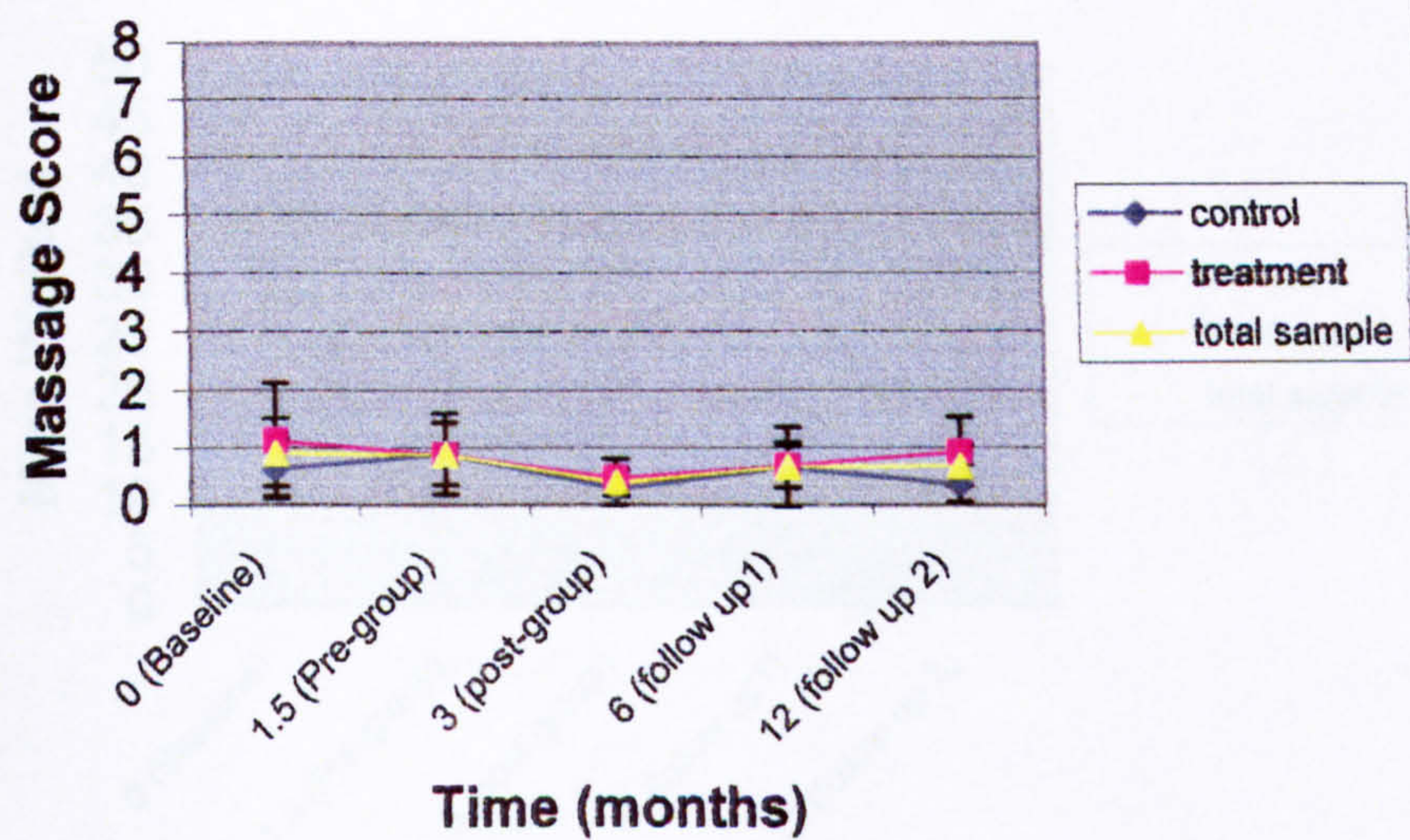


Figure 7.48: Graph showing control, treatment & total sample mean & 95% confidence intervals for frequency of walks between time 1 & 5

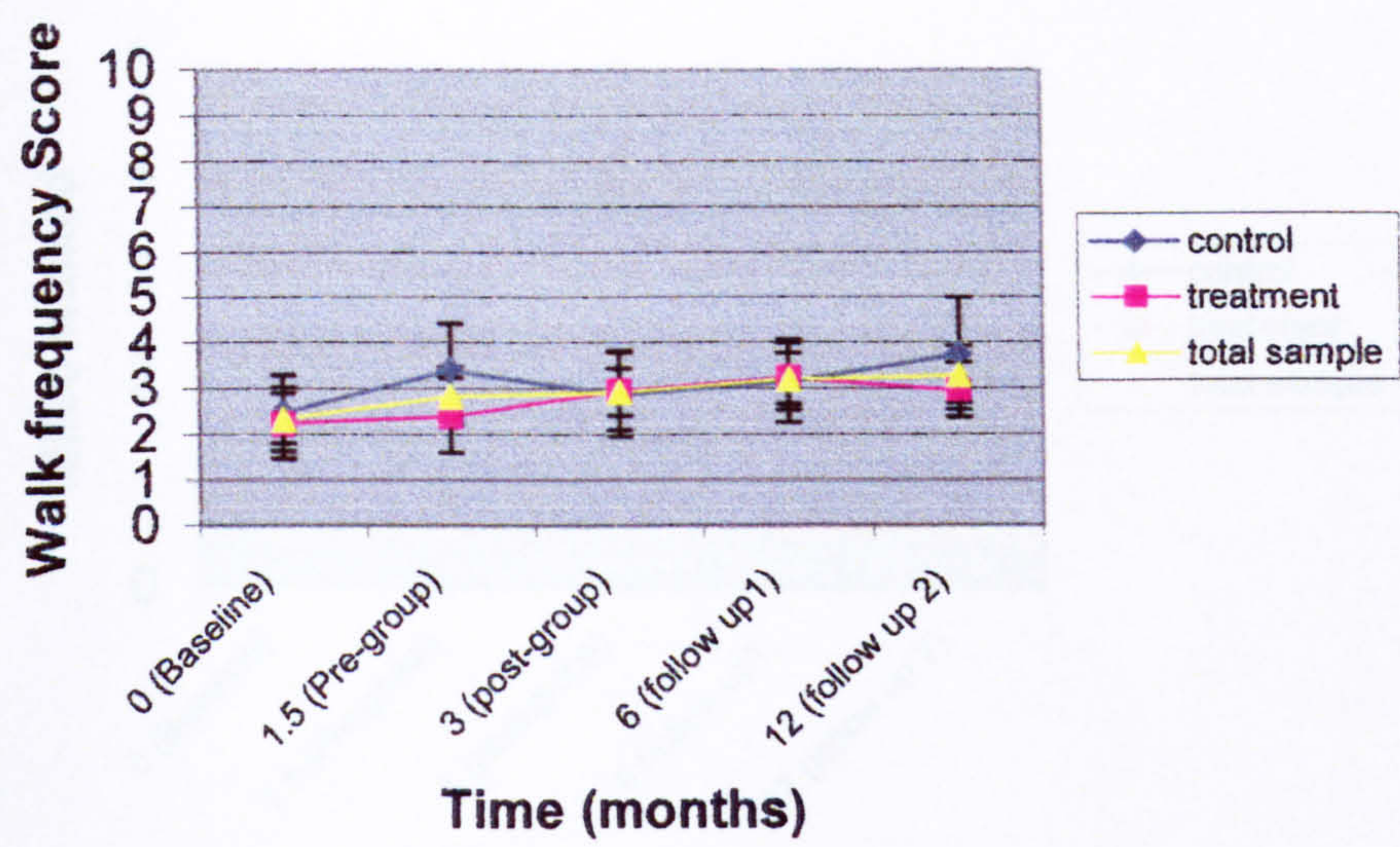


Figure 7.49: Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins walked between time 1 & 5

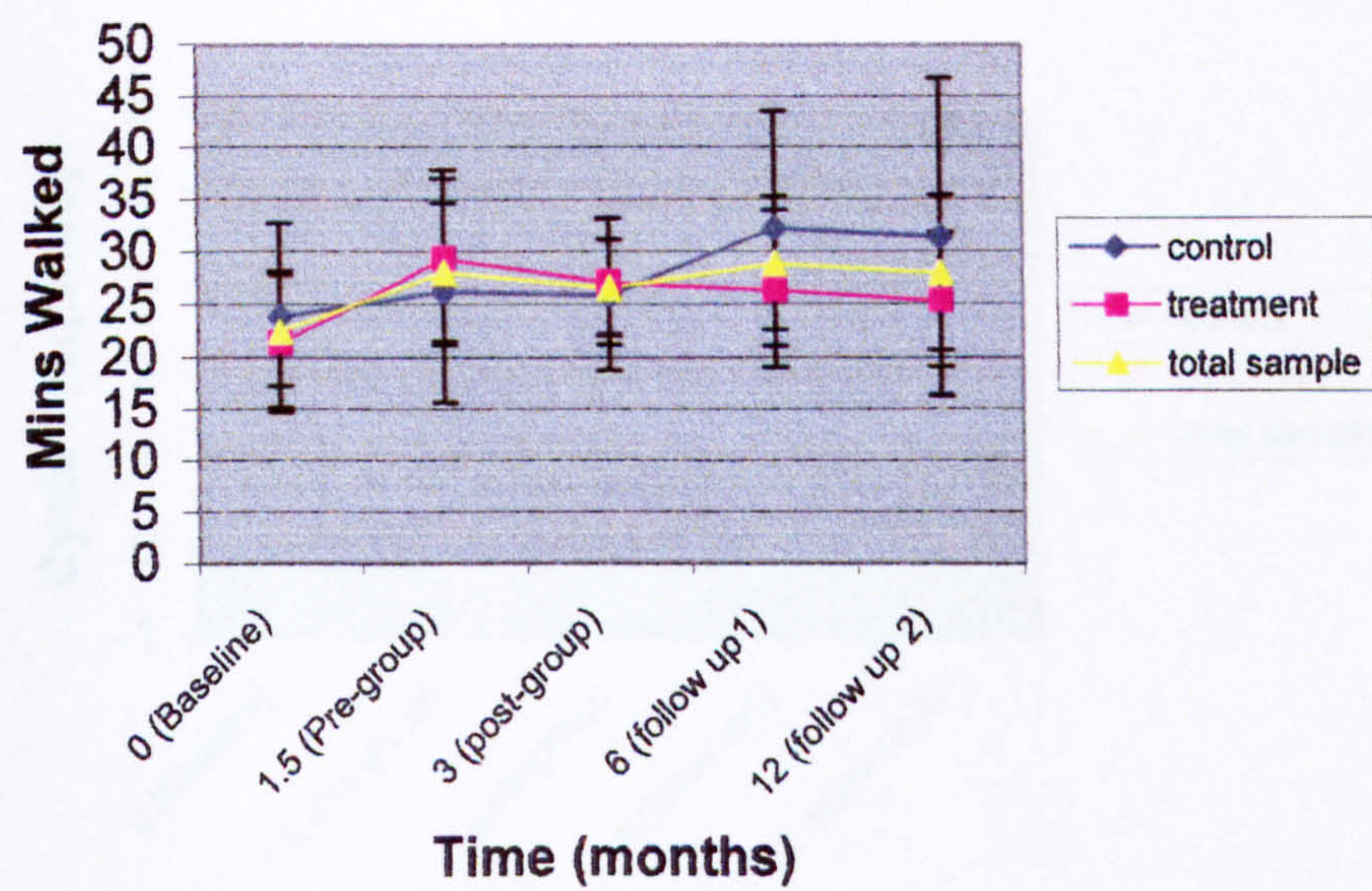


Figure 7.50: Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of miles walked between time 1 & 5

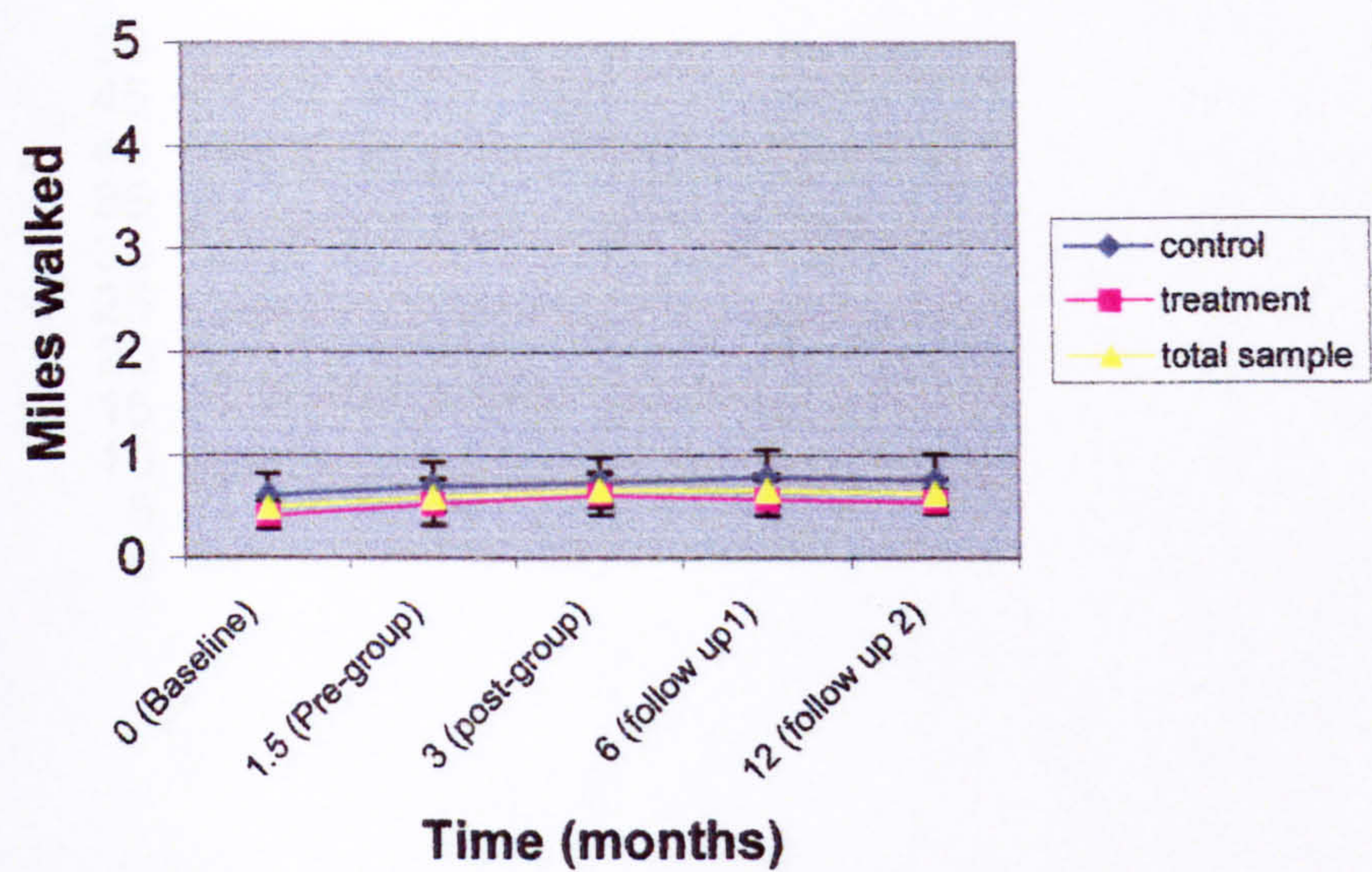


Figure 7.51: Graph showing control, treatment & total sample mean & 95% confidence intervals for frequency of cycling between time 1 & 5

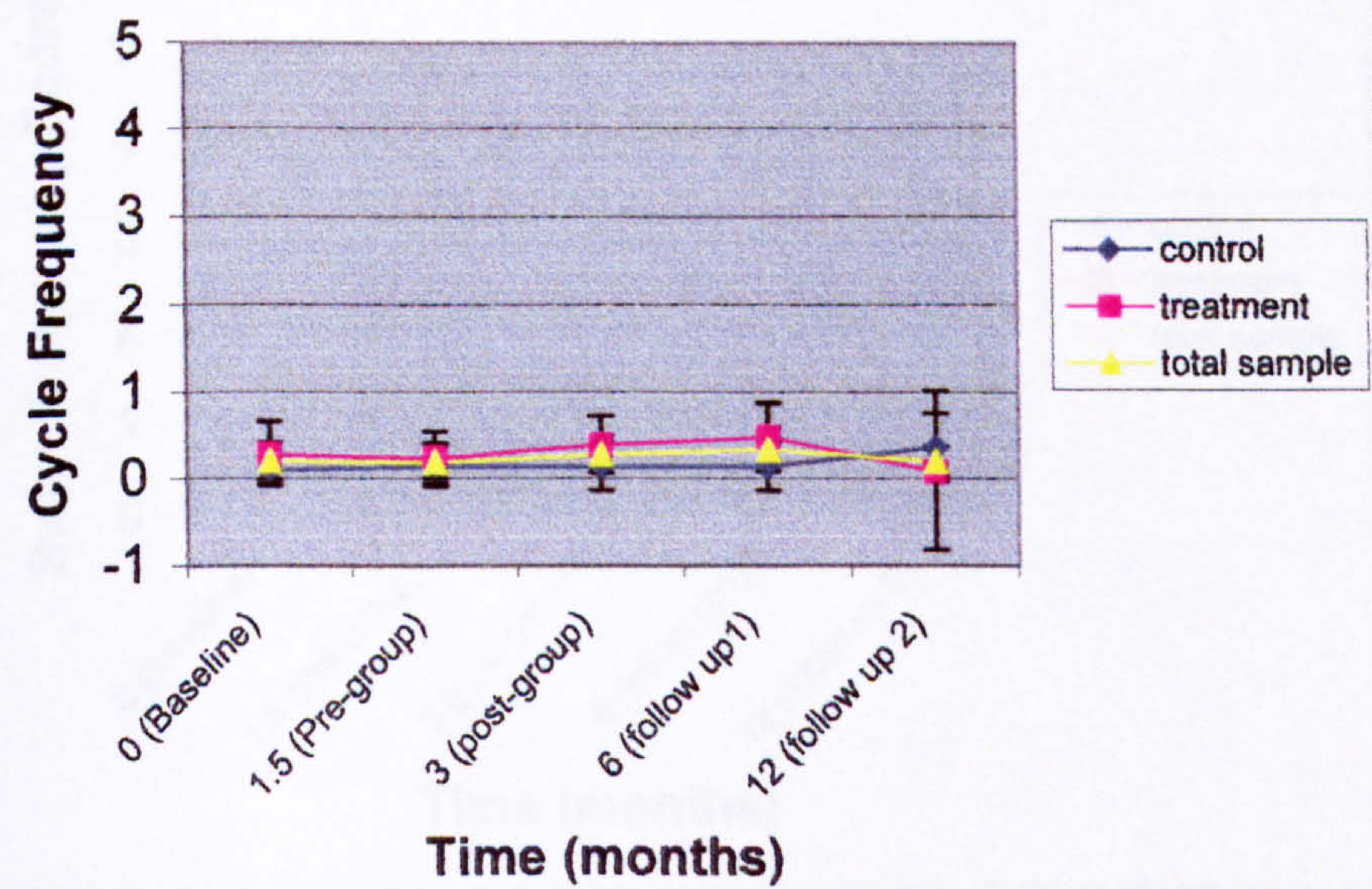


Figure 7.52: Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins cycled between time 1 & 5

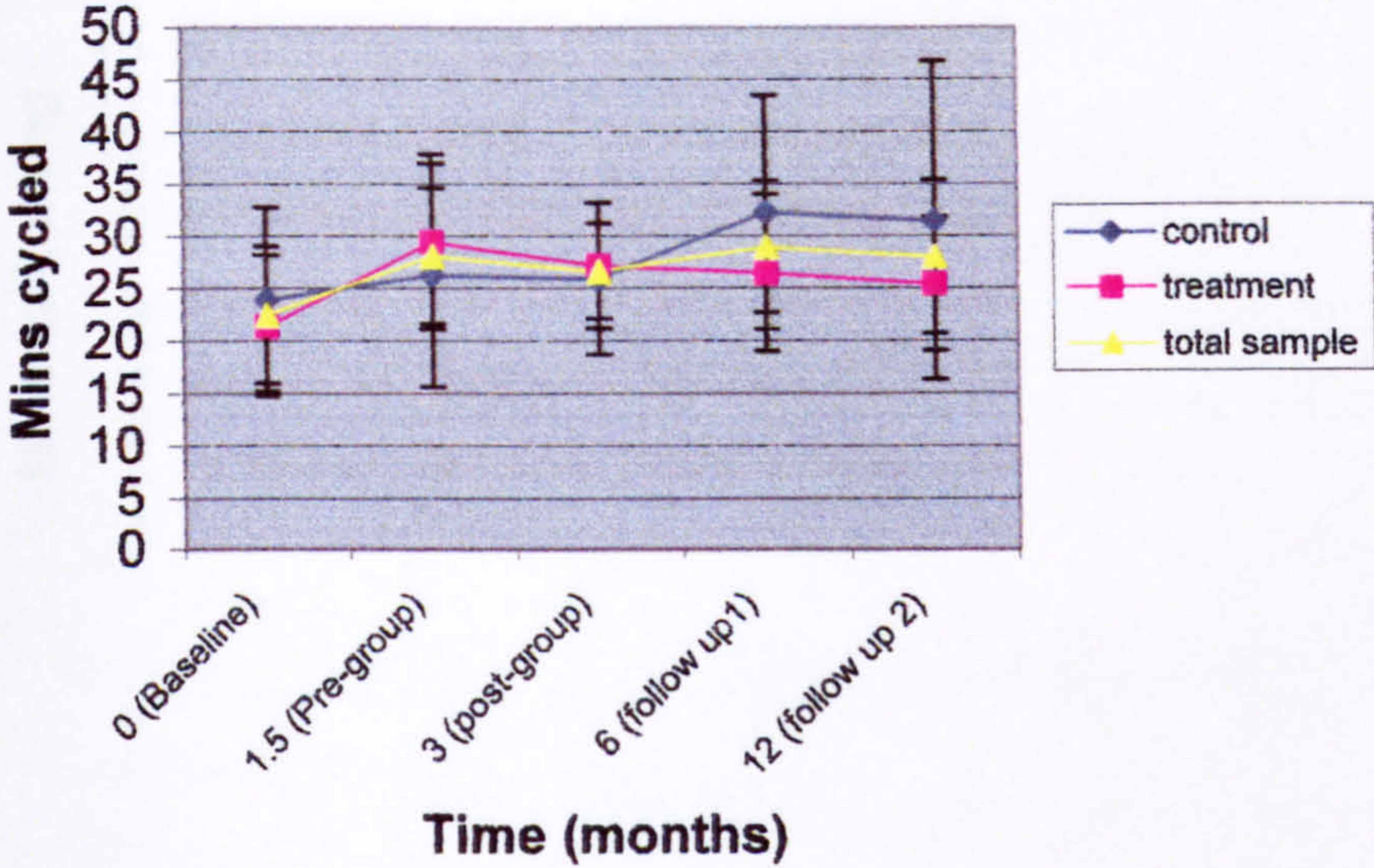


Figure 7.53: Graph showing control, treatment & total sample mean & 95% confidence intervals for frequency of swimming scores between time 1 & 5

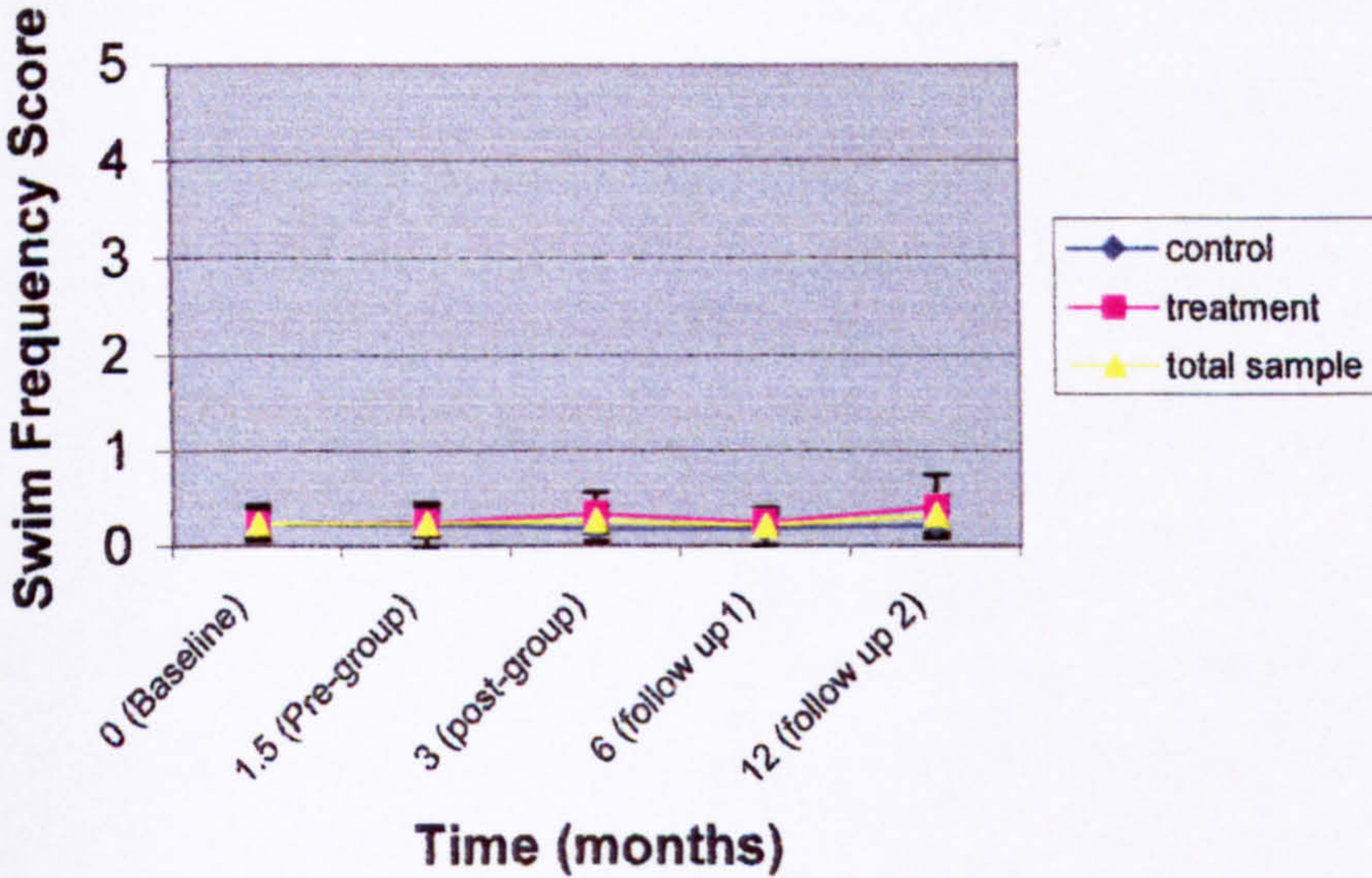
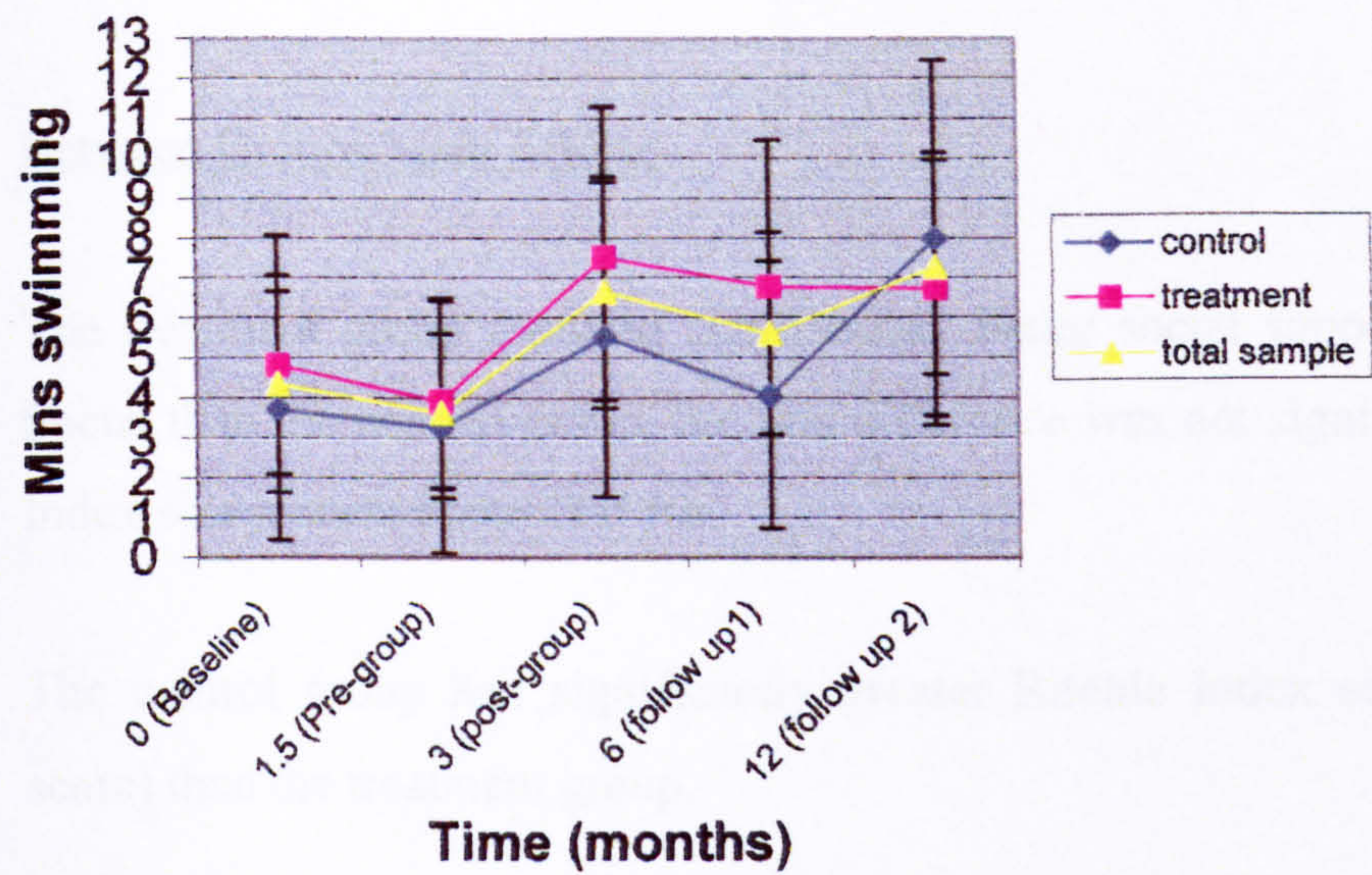


Figure 7.54: Graph showing control, treatment & total sample mean & 95% confidence intervals for No. of mins swimming scores between time 1 & 5



7.4 Summary of findings

7.4.1 Research question 1.4 and hypothesis 1.1 summary Of findings

Between Groups Main Effects

The treatment group reported significantly better social support and activity on AIMS Social than the control group, but this difference was not significant when initial Ritchie Index scores were controlled for.

The control group had significantly greater Ritchie Index scores (greater tender joint score) than the treatment group.

Within Participants Main Effects – Research Question 1.4

The significant within participants main effects answer Research Question 1.4 “How do health outcomes in a sample of people with RA change over one year?”

When change in disease activity (CRP) during the study was controlled for there was a significant improvement in emotional well being in the whole sample measured on AIMS2 Affect.

There was a significant improvement in quality of life (PGI) for the whole sample over the study. Most of the improvement occurs in the treatment group between time 1 and 3. When initial Ritchie Index scores are controlled for the result is no longer significant.

Over the study there was a significant improvement in the tender joint score measured on the Ritchie Index for both groups. Most of the change occurred in the first three months between time 1 and 3.

There was a significant reduction in disease activity measured on the CRP for the whole sample across the course of the study, much of the reduction occurred between time 1 and 2. This was not significant when initial Ritchie Index scores were controlled for.

Over the study there was a significant improvement (increase) in Haemoglobin levels (Hb) for the whole sample. Most of the change occurred during the first six months of the study. This was not significant when differences between the groups on Ritchie Index scores were controlled for in the ANCOVA.

There was a significant reduction in the frequency of doctor visits over the course of the study for the whole sample. Most of the reduction occurred between time 1 and 4 and was maintained at time 5. When initial Ritchie Index scores were controlled for the result was no longer significant.

Interaction Effects – Hypothesis 1.1

On the HAQ there was a significant interaction effect ($f = 2.57$, $d.f. = 3.607$, $p = 0.044^*$) indicating a difference between the treatment and control groups over time in their level of physical disability. Over the course of the study there is a small but non-significant improvement in both groups HAQ scores. Comparing the two groups it is clear that there is a larger improvement in the treatment group from time 1-2 but the difference is gradually reversed over the course of the study. The treatment group shows a significantly greater improvement in their HAQ scores but this occurs before the treatment. The difference between the treatment and control groups disappears by 12 months and when differences in initial RI and CRP change are controlled for. Overall this does not provide any support for hypothesis 1.1.

On the PGI there was a significant interaction effect ($f = 2.674$ $df = 3.759$ $p = 0.035^*$) indicating a difference between the treatment and control group in their PGI scores over time. Over the study the treatment group improved significantly more than the control group in quality of life measured on the PGI. This supports hypothesis 1.1 e that the

intervention will produce a significant improvement in quality of life. However, some of the improvement occurred during the baseline phase.

An interesting pattern of short-term results emerges from the ANOVA planned contrasts between time 1 and 3. There were reductions in pain (GRSP and AIMS Symptoms), disability (HAQ) and depression (HADS) for the whole sample shown by significant within participant main effects. Inspection of the graphs showed that most of the change occurred in the treatment group, especially between time 1 and 2. This was confirmed by significant interaction effects for GRSP and HAQ and trends towards significant interaction effects for AIMS Symptoms and HADS Depression between time 1 and 3. This suggests that the treatment group shows short-term improvements in pain, disability and depression immediately following the intervention. However, these improvements are unlikely to be fully accounted for by the intervention as much of the change occurs during the baseline phase. These changes might be accounted for by differences between the treatment and control groups Ritchie Index scores at baseline or change in CRP over time.

In summary the results provide little support for hypothesis 1.1 “The intervention will produce a significant improvement in the following health outcomes immediately after intervention, at 3 months and 9 month follow up.”

7.4.2 Research question 1.5 and hypothesis 1.2 summary of findings

Between Groups Main Effects

There were a number of differences between the groups shown by significant between groups effects on the ANOVA and/or ANCOVA.

The ANOVA showed that the treatment group had significantly higher perceived control/cure beliefs throughout the study measured on the IPQ. The difference was not significant when initial Ritchie Index scores were controlled for.

The treatment group reported significantly more use of the coping strategies seeking emotional social support, seeking instrumental social support, suppression of competing activities and restraint coping than the control group.

The treatment group report greater practice of stretching and strengthening exercises.

Within Group Main Effects – Research Question 1.5

For the whole sample there were changes over time on a number of measures shown by significant within participants main effects on the ANOVA and/or ANCOVA.

On the ANOVA there was a significant improvement in IPQ Coherence (less puzzling/distressing symptoms). The majority of the improvement occurred between time 1 and 3. The improvement is maintained at time 4 and 5.

On the ANCOVA there was a significant within participants effect for IPQ time line when controlling for initial Ritchie Index scores. However this is difficult to interpret.

On the ANOVA there was a significant increase in perceived self-efficacy for pain. The majority of the improvement was between time 2 and 3 in the treatment group. It was not significant when initial Ritchie index scores were controlled for in the ANCOVA.

The ANOVAs showed a significant reduction in the reported use of the coping strategies seeking emotional social support, seeking instrumental social support and focusing on and venting emotions. The change in seeking emotional social support and focusing on and venting emotions were not significant on the ANCOVA when initial Ritchie Index scores were controlled for.

There was a significant increase in the practice of stretching exercises, strengthening exercises and relaxation for the whole sample. Most of the improvement occurred immediately following treatment. The treatment group showed large increases in their

practice of these self-management behaviours but the control group also showed some improvement. The treatment group's improvements were also maintained, to some extent, at time 4 and 5. This provides some evidence that the intervention increases practice of self-management behaviours. However, as the control group also improves to some extent, the difference between the groups is not sufficient to produce a significant interaction effect on the main ANOVA. However for strengthening exercises, there was a significant interaction effect on the planned contrasts between time 2-3. This indicates that the treatment group improved significantly more than the control group following the intervention.

On the ANOVA there was a significant increase in the number of walks over half a mile per week and the number of miles walked per week for the whole sample. This was not significant on the ANCOVA when initial Ritchie Index scores were controlled for.

When initial Ritchie Index scores were controlled for in the ANCOVA there was a significant increase in the number of minutes cycling per week reported by the whole sample. There was a significant increase in the number of minutes per week swimming for the whole sample. This was not significant on the ANCOVA when initial Ritchie Index scores were controlled for. The treatment group reported swimming for longer following the intervention and this was maintained at follow up.

With respect to Research Question 1.5, there was consistent evidence of change in illness representations and coping procedures in the whole sample over time. The following changes were found: decreased IPQ Coherence (less puzzling/distressing); decreased use of seeking instrumental social support as a coping strategy, increased practice of stretching exercises, strengthening exercises and relaxation.

Interaction Effects – Hypothesis 1.2

There were significant differences between the treatment and control groups over time shown by significant interaction effects on the ANOVA and/or ANCOVA.

There was a significant increase in perceived self-efficacy for pain in the treatment group but not the control group, shown by a significant interaction effect on the main ANOVA. The improvement occurred following the intervention and was maintained at the three and nine month follow-ups. The control group showed little change over time.

There was a significant increase in perceived self-efficacy for other arthritis symptoms in the treatment group and a decrease in the control group, shown by a significant interaction effect on the main ANOVA. The improvement occurred following the intervention and was maintained at the three and nine month follow-ups.

There was a significant difference between the treatment and control groups in their use of planning as a coping strategy over the course of the study, shown by a significant interaction effect on the ANOVA and ANCOVA. There was a significant increase in the use of planning in both groups at time 3, following the intervention, shown by a significant within groups main effect for the planned contrast time 2 vs 3. However, the treatment group shows a much larger increase, indicated by the significant interaction effect for the planned contrast time 1 vs 3. The treatment group's increase is maintained at time 4 but not at time 5, shown by significant interaction effects in the planned contrast for time 1 vs 4 but not for time 1 vs 5. At time 5 the treatment group mean is slightly greater than the baseline and the control group mean is slightly lower than the baseline. Overall this suggests that the intervention leads to greater use of planning as a coping strategy, and this is maintained at the three month follow up but not at the nine month follow up.

There was a significant difference between the treatment and control groups in their use of humour as a coping strategy over the course of the study. Over the study the treatment group reports increased use of humour as a coping strategy and the control group reports decreased use of humour. Most of the difference between the groups occurs during the baseline phase, time 1-2, before the intervention. This was not significant when initial

Ritchie Index scores were controlled for. This result does not suggest that the intervention produces a significant increase in the use of humour as a coping strategy.

Although the interaction effects for the main ANOVAs for IPQ Identity and IPQ Consequences were not significant, there were significant interaction effects found in the planned contrasts. For IPQ Identity, significant interaction effects, for time 1 vs 2, 1 vs 3 and 1 vs 4, indicate that the treatment group had significant decreases in IPQ Identity between time 1 and 3 which were maintained at time 4. For IPQ Consequences, significant interaction effects, for time 2 vs 3 and time 1 vs 3, indicate that the treatment group showed a significant decrease in perceived consequences following the intervention but that this was not maintained at time 4 or 5.

In summary, these results only partially support hypothesis 1.2 as the intervention group shows significantly greater adaptive changes in process variables than the control group. Immediately following the intervention the treatment group had significantly greater decreases in IPQ Identity and IPQ consequences, and increases in self-efficacy for pain, self-efficacy for other arthritis symptoms, use of planning as a coping strategy and practice of strengthening exercises, than the control group. The changes in IPQ Identity, self-efficacy for pain, self-efficacy for other arthritis symptoms and use of planning as a coping strategy were maintained at 3-month follow up. The improvements in self-efficacy for pain and other arthritis symptoms were also maintained at 9-month follow up.

Chapter 8: The relationship between illness representations, coping and health outcomes

8.1 Introduction

This chapter presents the results of analyses, in the pooled sample, exploring the relationships between self-regulatory processes and health outcomes in chronic RA. Section 8.3 presents the results of analyses examining the relationships between illness representations and coping procedures at baseline (hypothesis 2.1). Section 8.4 presents the results of the cross-sectional analyses, testing whether illness representations and coping procedures account for significant variance in health outcomes at baseline (Hypothesis 2.2). Section 8.5 presents the results of the longitudinal analyses, testing whether initial illness representations and coping procedures predict change in health outcomes at 3, 6 and 12 months (Hypothesis 2.3). Section 8.6 presents the results of the longitudinal analyses, testing whether changes in illness representations and coping procedures predict change in health outcomes at 3, 6 and 12 months (Hypothesis 2.4). For clarity, summaries of the findings, for each hypothesis, are presented at the end of the relevant section, rather than at the end of the chapter.

8.2 Statistical methods

To test hypothesis 2.1 a series of Pearson product moment correlation coefficients were calculated to examine relationships between illness representation and coping variables.

Testing hypotheses 2.2, 2.3 and 2.4 involved performing a series of Hierarchical multiple regression analyses. For all these analyses the data was assessed to ensure it met the assumptions of linear regression, with Kolmogorov-Smirnoff one-sample tests used to test the normality of the residual deviations from the regression analyses. The analysis was rejected if Z was significant at the $p=0.05$ level. In addition scatter plots were undertaken of residuals against predicted values to test the assumption of constant variance. Independent variables were usually only entered in the regression if they were significantly correlated with the dependent variable. Variables that measured the same construct as the dependent variable were not included in the regression to eliminate strong reciprocal relationships between measures. When the dependent variables were pain measures the multiple regression analyses were computed twice. First, including IPQ Identity and IPQ Coherence as independent variables, second, excluding IPQ Identity from all analyses and IPQ Coherence from the analyses of pain distress. This approach was used because it is possible that strong reciprocal relationships exist between pain measures and these variables. However, it is clear that IPQ Identity measures broader concepts than a pain intensity scale, reflecting people's perception of the range and frequency of different symptoms that they attribute to their RA. IPQ Coherence also measures broader concepts than a pain distress measure, as it assesses the extent to which people perceive all their RA symptoms as puzzling, changeable and distressing.

To test Hypothesis 2.2 a series of Hierarchical multiple regression analyses were performed. As this is a cross-sectional analysis it included time 1, baseline, values for both dependent and independent variables.

Independent variables were entered in blocks in the following order:

1. Experimental condition (0 = control 1 = treatment) this was entered even if it was not significantly correlated with the dependent variable.
2. Socio-demographic, and disease and disability variables.
3. Emotional distress variables

4. Illness representations, coping, self-management and self-efficacy variables.

In the hierarchical multiple regression analyses, used to test hypotheses 2.3 and 2.4, the regression model was set up so that for each dependent variable hypothesis 2.3 and 2.4 could be tested in the same analysis. Independent variables were entered in blocks in the following order:

- 1. Experimental condition (0 = control 1 = treatment) this was entered even if it was not significantly correlated with the dependent variable.**
- 2. The baseline (time 1) score for the dependent variable.**
- 3. Socio-demographic, and baseline disease, disability and social function variables.**
- 4. Baseline emotional distress variables**
- 5. Baseline (time 1) illness representations, coping, self-management and self-efficacy variables (Hypothesis 2.3). Time 1 scores for any variables entered in step 6 were included even if not significantly correlated with the dependent variable to allow the testing of Hypothesis 2.4 in the next step. This controls for the baseline level of the independent variables entered in step 6, which are concurrent with the dependent variable. This avoids the problems associated with analyses of change scores.**
- 6. Illness representations, coping, self-management and self-efficacy variables time 3, 4 or 5 (Hypothesis 2.4).**

8.3 The relationship between illness representations and coping procedures

Hypothesis 2.1 At baseline the following illness representations will be positively associated with greater use of adaptive coping (*acceptance, active coping, positive reinterpretation/growth and planning*) and less use of maladaptive coping (*behavioural and mental disengagement, denial and focusing on and venting emotions*):

- a. Lower illness identity
- b. Greater symptom coherence
- c. Less serious perceived consequences
- d. More chronic timeline
- e. Greater perceived controllability
- f. Less perceived causes

The results of the correlational analyses are presented in Table 8.1. As predicted IPQ identity was significantly negatively correlated with positive reinterpretation and growth and positively correlated with behavioural disengagement and denial. People who identified fewer symptoms as part of their RA made greater use of the adaptive coping strategy Positive Reinterpretation/Growth and less use of the maladaptive strategies, Behavioural Disengagement and Denial.

As predicted IPQ Coherence was significantly negatively associated with Positive Reinterpretation/Growth and positively associated with Behavioural Disengagement. People who perceived their RA symptoms as more coherent (less puzzling, changeable and distressing) made greater use of the adaptive coping strategy Positive Reinterpretation/Growth and less use of the maladaptive strategy Behavioural Disengagement.

As predicted IPQ Consequences was significantly negatively associated with Positive Reinterpretation/Growth and Acceptance and positively associated with Focusing on and

venting emotions and Behavioural Disengagement. People who perceived RA as having less serious consequences made greater use of adaptive coping strategies, Positive Reinterpretation/Growth and Acceptance, and less use of maladaptive strategies, Focusing and venting emotions and Behavioural Disengagement.

IPQ Time Line was not significantly associated with any coping strategies.

IPQ Control/Cure was positively associated with Active Coping and Planning. People who perceived RA as more controllable made greater use of adaptive coping strategies, Active Coping and Planning.

IPQ Total Attributions was not significantly associated with the use of any coping strategies.

In summary there is partial support for Hypothesis 2.1 a, b, c and e. All significant relationships were in the predicted directions although not all predicted relationships were found to be significant. An interesting pattern emerged. Identifying fewer symptoms as part of RA, perceiving RA symptoms to be more coherent and the consequences of RA to be less serious were all associated with: 1. greater use of the adaptive coping strategy, Positive Reinterpretation/Growth; 2. less use of the maladaptive strategy, Behavioural Disengagement. The strongest relationships were found between Identity, Coherence and Consequences and Behavioural Disengagement ($r=-0.24$ to -0.43).

Table 8.1: Correlations of time 1 IPQ and cope variables

	Cope: Positive Interpret/growth	Cope: Active Coping	Cope: Planning	Cope: Acceptance	Cope: Mental Disengagement	Cope: Focusing/venting emotions	Cope: Behavioural disengagement	Cope: Denial
IPQ Identity	-0.18*	-0.10	-0.02	-0.1	-0.02	0.15	0.43** *	0.23*
IPQ Coherence	-0.20*	0.08	0.09	-0.17	0.06	0.12	0.24**	0.18
IPQ Consequences	-0.21*	-0.06	-0.17	-0.19*	0.08	0.19*	0.31**	0.13
IPQ Timeline	-0.17	-0.01	-0.16	0.18	0.10	0.12	0.11	0
IPQ Control/Cure	0.08	0.23*	0.18*	-0.11	0.01	-0.01	-0.15	-0.18
IPQ Total Attribution	-0.03	0.03	0.11	-0.10	-0.06	0.09	-0.12	0.01

* = $p < 0.05$, ** = $p < 0.01$, *** $p < 0.001$

8.4 The relationship between illness representations and coping procedures, and health outcomes at baseline

Hypothesis 2.2 Illness representations and coping procedures will account for a significant amount of variance in the following health outcomes in the pooled sample (independent of that accounted for by experimental condition) at time 1.

- a Pain
- b Physical disability
- c Emotional distress
- d Social/Occupational Function
- e Quality of life (PGI)
- f Disease activity
- g Health Care utilization

This hypothesis explores Leventhal's self-regulatory model which proposes that illness representations shape coping procedures which in turn influence health outcomes. The analyses assess the extent to which illness representation, coping, self-management and self-efficacy accounted for health outcomes cross-sectionally at time 1. The results of the cross-sectional multiple regression analyses are described below and summarized in Tables 8.2-8. The tables include the total amount of variance in the dependent variable explained by the final model (Adjusted R^2 % variance) and the additional variance explained at each significant step in the analysis (Adjusted R^2 % change; $\Delta adj.R^2$). The tables also include the significant predictors, from the final model, presented under the column representing the step at which they were first entered in the analysis. For each significant predictor the beta weight (β), and unique variance explained (r_{sp}^2) are also presented in the tables and text.

8.4.1 Explaining baseline pain

The cross-sectional multiple regression analyses explaining pain are shown in Table 8.2.

NRIP

The regression explained 36.1% of the variance in pain (NRIP) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 6.6% variance. In the final model, lower pain was predicted by higher disability, AIMS2 Physical (-0.49**; 0.04); higher positive affect, PANAS (-0.24*; 0.02); lower IPQ Identity (0.3*; 0.03); lower self-efficacy for other arthritis symptoms (0.3*; 0.03) and higher self-efficacy for function (-0.45*; 0.05).

The findings for AIMS2 Physical and self-efficacy for other arthritis symptoms are unexpected. The correlation between NRIP and AIMS2 Physical ($r=0.29^{**}$) and self-efficacy for other arthritis symptoms ($r=0.3^{**}$) are positive. However, when all variables in the regression have been partialled out what remains is a negative relationship. This result may reflect a degree of overlap between the independent variables or a suppression effect.

GRSP

The regression explained 30.6% of the variance in pain (GRSP) at time 1. When entered, in model 4, illness representations and coping procedures explained a significant additional 6.2% variance. In the final model, lower pain was predicted by higher disability, AIMS2 Physical (-0.38*; 0.03); lower negative affect, PANAS (0.31*; 0.03); higher self-efficacy for function (-0.38*; 0.03).

BVAS

The regression explained 32.5% of the variance in pain (BVAS) at time 1. When entered, in model 4, illness representations and coping procedures explained an additional (5.4%)

variance, but the model was not significant. In the final model there were no significant predictors. There are also no significant predictors in model 3, which was the last significant model.

Pain distress

The regression explained 37.8% of the variance in pain distress at time 1. When entered in model 4, illness representations and coping procedures explained an additional (4.8%) variance, but the model was not significant. In the final model, lower pain distress was predicted by higher self-efficacy for function (-0.37*; 0.03). In model 3, which was significant, lower pain was predicted by lower negative affect, PANAS (0.34*; 0.04).

AIMS2 Symptoms

The regression explained 48.1% of the variance in pain (AIMS2 Symptoms) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 13.3% variance. In the final model, lower pain was predicted by higher use of acceptance as a coping strategy (-0.18*; 0.02); lower IPQ Identity (0.3*; 0.03).

Table 8.2: Summary of cross sectional multiple regression analyses explaining pain at time 1 (IPQ Identity and Coherence included in analyses).

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)	1	2	3
			Experimental condition	Demographics Disease	Emotional
NRIP T1	36.1%			13.9% AIMS2 Physical (-0.49**; 0.04)	13.4% PANAS Positive Affect (-0.24*; 0.02)
					6.6% IPQ Identity (0.30*; 0.03) Self-efficacy: Function (-0.45*; 0.05) Self-efficacy: Arthritis (0.3*; 0.03)
GRSP T1	30.6%			10.8% AIMS2 Physical (-0.38*; 0.03)	13.6% PANAS Negative Affect (0.31*; 0.03)
					6.2% Self-efficacy: Function (-0.38*; 0.03)
BVAS T1	32.5%			14.3%	11.9%
					5.4% (NS)
Pain Distress T1	37.8%			17.4%	15.6%
					4.8% (NS) Self-efficacy: Function (-0.37*; 0.03)
AIMS2 Symptoms T1	48.1%			25.2%	9.6%
					13.3% Cope: Acceptance (-0.18*; 0.02) IPQ Identity (0.3*; 0.03)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.2 Explaining baseline pain (excluding IPQ Identity and IPQ Coherence)

The multiple regression analyses for pain were also computed excluding IPQ Identity from all analyses and IPQ Coherence from the analysis where pain distress was the dependent variable. The cross-sectional multiple regression analyses explaining pain (excluding IPQ Identity and IPQ Coherence) are shown in Table 8.3.

NRIP

The regression explained 33.3% of the variance in pain (NRIP) at time 1. When entered in model 4, illness representations and coping procedures explained an additional (6.7%) variance, but the model was not significant. In the final model, lower pain was predicted by higher disability AIMS2 Physical (-0.44*; 0.03); higher self-efficacy for pain (-0.24*; 0.04) and higher self-efficacy for function (-0.43**; 0.03). In model 3, the last significant model lower pain was predicted by lower disability HAQ (0.34*; 0.03); higher disability AIMS2 Physical (-0.34*; 0.03); lower tender joint score Ritchie (0.22*; 0.04); higher positive affect, PANAS (-0.27*; 0.03).

GRSP

The regression explained 29.2% of the variance in pain (GRSP) at time 1. When entered in model 4, illness representations and coping procedures explained an additional (4.8%) variance, but the model was not significant. In the final model, lower pain was predicted by higher disability AIMS2 Physical (-0.38*; 0.03); lower negative affect, PANAS (0.32*; 0.03); lower depression POMS dejection/depression (-0.3*; 0.03); higher self-efficacy for function (-0.35*; 0.03). In model 3, the last significant model, lower pain was predicted by lower disability HAQ (0.34*; 0.03); lower negative affect, PANAS (0.31*; 0.03). higher positive affect PANAS (-0.27*; 0.03).

BVAS

The regression explained 30.3% of the variance in pain (BVAS) at time 1. When entered, in model 4, illness representations and coping procedures explained an additional (3.2%) variance, but the model was not significant. In model 4, there were no significant predictors and in model 3, the last significant model, there were no significant predictors.

Pain distress

The regression explained 35.9% of the variance in pain distress at time 1. When entered in model 4, illness representations and coping procedures explained an additional (2.9%) variance, but the model was not significant. In the final model, lower pain was predicted by lower negative affect, PANAS (0.31*; 0.03). In model 3, the last significant model, lower pain was predicted by lower negative affect PANAS (0.34*; 0.04).

AIMS2 Symptoms

The regression explained 45.1% of the variance in pain (AIMS2 Symptoms) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 10.3% variance. In the final model, lower pain was predicted by higher self-efficacy for pain (-0.26*; 0.03) and greater use of acceptance as a coping strategy (-0.19*; 0.02).

Table 8.3: Summary of cross sectional multiple regression analyses explaining pain at time 1 (excluding IPQ Identity and IPQ Coherence).

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)	1	2	3
			Experimental condition	Demographics Disease	Emotional
NRIP T1	33.3%			13.6% AIMS2 Physical (-0.44*; 0.03)	13%
					6.7% (NS) Self-efficacy: Pain (-0.24*; 0.04) Self-efficacy: Function (-0.43**; 0.03)
GRSP T1	29.2%			10.8% AIMS2 Physical (-0.38*; 0.03)	13.6%
					PANAS Negative Affect (0.32*; 0.03) POMS Depression/dejection (-0.3*; 0.03)
BVAS T1	30.3%			14.3%	11.9%
					3.2% (NS)
Pain Distress T1	35.9%			17.4%	15.6%
					PANAS Negative Affect (0.31*; 0.03)
AIMS2 Symptoms T1	45.1%			25.2%	9.6%
					10.3% Self-efficacy: Pain (-0.26*; 0.03) Cope: Acceptance (-0.19*; 0.02)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.3 Explaining baseline physical disability

The cross-sectional multiple regression analyses explaining physical disability are shown in Table 8.4.

HAQ

The regression explained 70.9% of the variance in disability (HAQ) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 51.8% variance. In the final model, lower disability was explained by lower POMS fatigue/inertia (0.25*; 0.02); higher self-efficacy for function (-0.80***; 0.29); less use of behavioural disengagement as a coping strategy (0.25***; 0.03) and lower self-efficacy for other arthritis symptoms (0.25*; 0.02).

The finding for self-efficacy for other arthritis symptoms is unexpected. The correlation between HAQ and self-efficacy for other arthritis symptoms ($r=-0.38^{**}$) is negative. However, when all variables in the regression have been partialled out what remains is a positive relationship. This result may reflect a degree of overlap between the independent variables or a suppression effect.

AIMS2 Physical

The regression explained 71.4% of the variance in disability (AIMS2 Physical) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 26.3% variance. In the final model, lower disability was explained by lower tender joint score Ritchie (0.15*; 0.01); higher self-efficacy for function (-0.54***; 0.11) and lower use of focusing on and venting emotions as a coping strategy (0.21*; 0.02).

Table 8.4: Summary of cross sectional multiple regression analyses explaining disability at time 1.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)			
		1 Experimental condition	2 Demographics Disease	3 Emotional	4 Cognitions and Coping
HAQ T1	70.9%		22.1%	POMS Fatigue/Inertia (0.25*; 0.02)	51.8% Self-efficacy: Function (-0.80***; 0.29) Cope: Behavioural Disengagement (0.25***; 0.03) Self-efficacy: Arthritis (0.25*; 0.02)
AIMS2 Physical T1	71.4%		37.8% Ritchie (0.15*; 0.01)	7.3%	26.3% Self-efficacy: Function (-0.54***; 0.11) Cope: Focus/Vent Emotions (0.21*; 0.02)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.4 Explaining baseline emotional distress

The cross-sectional multiple regression analyses explaining emotional distress are shown in Table 8.5.

HADS Depression

The regression explained 66% of the variance in depression (HADS Depression) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 26.3% variance. In the final model, lower depression was predicted by better social function, AIMS2 Social (0.18*; 0.02); perceiving the consequences of RA as less serious, IPQ Consequences (0.27**; 0.03); less use of suppression of competing activities as a coping strategy (0.22**; 0.03) and less use of behavioural disengagement as a coping strategy (0.17*; 0.02).

HADS Anxiety

The regression explained 60.3% of the variance in anxiety (HADS Anxiety) at time 1. When entered, in model 4, illness representations and coping procedures explained a significant additional 29.1% variance. In the final model, lower anxiety was predicted by higher disease activity, ESR (-0.29***; 0.06); lower IPQ Identity (0.36***; 0.05); perceiving symptoms of RA as less puzzling/distressing IPQ Coherence (0.18*; 0.02); perceiving arthritis as more controllable/curable (-0.23**; 0.03); greater use of acceptance as a coping strategy (-0.2**; 0.03); less use of behavioural disengagement as a coping strategy (0.17*; 0.02); less use of focusing on and venting emotions as a coping strategy (0.32***; 0.06).

The finding that lower disease activity (ESR) predicted higher anxiety is interesting. In the preliminary correlational analyses ESR was also found to be significantly negatively associated with HADS anxiety ($r=-0.24^*$). One possible explanation might be that in a chronic fluctuating illness like RA a period of time when the disease is less active may increase anxiety because it heightens awareness of the impact of the condition on the

person's life during periods of high disease activity and anticipation of future worsening in health. An alternative explanation might be that people with RA who have higher levels of anxiety may be more likely to be attending hospital out-patient clinics, even when their disease is relatively less active.

AIMS2 Affect

The regression explained 55.6% of the variance in emotional distress, (AIMS2 Affect) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 13.1% variance. In the final model, lower emotional distress was predicted by lower IPQ Identity (0.2*; 0.02); less use of focusing on and venting emotions as a coping strategy (0.31***; 0.05) and less use of behavioural disengagement as a coping strategy (0.19*; 0.02).

Table 8.5: Summary of cross sectional multiple regression analyses explaining emotional distress at Time 1.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)	1	2	3
			Experimental condition	Demographics Disease	Emotional
					4 Cognitions and Coping
HADS Depression T1	66%			39.7% AIMS2 Social (0.18*; 0.02)	26.3% Cope: Suppression of Competing Activities (0.22**; 0.03) Cope: Behavioural Disengagement (0.17*; 0.02) IPQ Consequences (0.27**; 0.03)
HADS Anxiety T1	60.3%			31.2% ESR (-0.29***; 0.06)	29.1% Cope: Acceptance (-0.2**; 0.03) Cope: Behavioural Disengagement (0.17*; 0.02) Cope: Focus/vent emotions (0.32***; 0.06) IPQ Control/Cure (-0.23**; 0.03) IPQ Identity (0.36***; 0.05) IPQ Coherence (0.18*; 0.02)
AIMS2 Affect T1	55.6%			42.5%	13.1% Cope: focus/vent emotions (0.31***; 0.05) Cope: behavioural disengagement (0.19*; 0.02) IPQ Identity (0.2*; 0.02)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.5 Explaining baseline social/occupational function

The cross-sectional multiple regression analyses explaining social and occupational function are shown in Table 8.6.

AIMS2 Social

The regression explained 28.9% of the variance in social function (AIMS2 Social) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 14.3% variance. In the final model, higher social function was predicted by older age (-0.25*; 0.03), greater self-efficacy for other arthritis symptoms (-0.34*; 0.02); higher IPQ Identity (-0.3*; 0.03) and greater use of seeking emotional social support as a coping strategy (-0.27*; 0.04).

The finding for IPQ Identity is unexpected. The correlation between AIMS2 Social and IPQ Identity is positive ($r=0.229^*$), perceiving a greater range and frequency of RA symptoms is associated with poorer social function. However, when all variables in the regression have been partialled out what remains is a negative relationship. This result may reflect a degree of overlap between the independent variables or a suppression effect.

AIMS2 Role

The regression explained 17.0% of the variance in occupational function (AIMS2 Role) at time 1. When entered in model 4, illness representations and coping procedures only explained 0.5% variance and this was not significant. In the final model, greater occupational function was predicted by lower disability HAQ (0.53**; 0.09); higher disability AIMS2 physical (-0.37*; 0.04) and having less children (0.22*; 0.04). In model 2, the last significant model, better occupational function was predicted by lower disability HAQ (0.54**; 0.10).

Table 8.6: Summary of cross sectional multiple regression analyses explaining social and occupational function at Time 1.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		Δ adj.R ² (β ; r_{sp}^2)	1	2	3
			Experimental condition	Demographics Disease	Emotional
					4 Cognitions and Coping
AIMS2 Social T1	28.9%			10.0% Age (-0.25*; 0.03)	14.3% Self-efficacy : Arthritis (-0.34*; 0.02) IPQ Identity (-0.3*; 0.03) Cope: Seeking Social Support Emotional (-0.27*; 0.04)
AIMS2 Role T1	17.0%			16.4% HAQ (0.53**; 0.09) Number of children (0.22*; 0.04) AIMS2 Physical (-0.37*; 0.04)	0.5% (NS)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.6 Explaining baseline individualized quality of life

The cross-sectional multiple regression analysis explaining individualized quality of life is shown in Table 8.7.

PGI

The regression explained 49.4% of the variance in individualized quality of life (PGI) at time 1. When entered in model 4, illness representations and coping procedures explained 7.5% variance, but the model was not significant. In the final model, higher individualized quality of life was predicted by perceiving the consequences of RA to be less serious, IPQ Consequences (-0.28*; 0.03). In model 2, the last significant model, there were no significant predictors.

Table 8.7: Summary of cross sectional multiple regression analyses explaining individualized quality of life at Time 1.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)			
		1 Experimental condition	2 Demographics Disease	3 Emotional	4 Cognitions and Coping
PGI T1	49.4%		37.2%		7.5% (NS) IPQ Consequences (-0.28*; 0.03)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.7 Explaining baseline disease activity

The cross-sectional multiple regression analyses explaining disease activity are shown in Table 8.8.

CRP

The regression explained 24.9% of the variance in disease activity (CRP) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 6.5% variance. In the final model, lower disease activity (CRP) was predicted by lower age (0.21*; 0.03); greater POMS confusion/bewilderment (-0.29**; 0.06) and identifying fewer causes for RA IPQ Cause Total Attributions (0.26**; 0.05).

The negative association between CRP and POMS: confusion/bewilderment is interesting and was also found in the correlational analysis ($r=-0.190^*$). There seems to be no obvious explanation for why people who have less active disease should feel more confused or bewildered and this finding may merit further exploration.

ESR

The regression explained 28.0% of the variance in disease activity (ESR) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 4.7% variance. In the final model, lower disease activity (ESR) was predicted by identifying fewer causes for RA IPQ Cause Total Attributions (0.23**; 0.05).

HB

The regression explained 4.8% of the variance in disease activity (HB) at time 1. No emotional, illness representation or coping variables were entered into the regression in model 3 or 4. In model 2, the final model, greater haemoglobin levels (lower disease activity) were explained by being male (-0.23*; 0.05). This reflects the normal difference found between males and females with RA.

WCC

The regression explained 8.7% of the variance in disease activity (white cell count) at time 1. When entered in model 4, illness representations and coping procedures only explained 1.0% variance and the model was not significant. In model 4 there were no significant predictors. In model 2, the last significant model, higher white cell count was predicted by higher disability AIMS2 physical (0.39*; 0.04).

Platelets

The regression explained 11.1% of the variance in disease activity (platelets) at time 1. When entered in model 4, illness representations and coping procedures explained a significant additional 6.6% variance. In the final model, lower platelets (less active disease) was predicted by identifying fewer causes for RA IPQ Cause Total Attributions (0.2*; 0.04) and less use of mental disengagement as a coping strategy (0.19*; 0.03).

Ritchie Index

The regression explained 21.2% of the variance in tender joint score (Ritchie) at time 1. When entered in model 4, illness representations and coping procedures only explained 1.3% variance and the model was not significant. In the final model, lower tender joint score was predicted by lower disability AIMS2 physical (0.44*; 0.04). In model 2, the last significant model, lower tender joint score was predicted by lower disability AIMS2 physical (0.32*; 0.03) and lower pain distress (0.20*; 0.03).

EMS

The regression explained 11.5% of the variance in early morning stiffness (EMS) at time 1. When entered in model 4, illness representations and coping procedures only explained 1.4% variance and the model was not significant. In the final model, a shorter duration of early morning stiffness was predicted by identifying fewer causes for RA IPQ Cause

Total Attributions (0.25*; 0.05). In model 3, the last significant model, early morning stiffness was predicted by greater POMS vigour/activity (-0.24*; 0.04).

Table 8.8 Summary of cross sectional multiple regression analyses explaining disease activity at Time 1.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES			
		$\Delta adj.R^2$ (β ; r_{sp}^2)	1	2	3
			Experimental condition	Demographics Disease	Emotional
CRP T1	24.9%			9.2% Age (0.21*; 0.03)	8.2% POMS: Confusion/Bewilderment (-0.29**; 0.06)
ESR T1	28.0%			7.6%	15.7%
					4.7% IPQ Cause (0.23**; 0.05)
HB T1	4.8%			4.4% Sex (-0.23*; 0.05)	
WCC T1	8.7%			7.7%	1.0% (NS)
Platelets T1	11.1%			2.1%	6.6% IPQ Cause (0.20*; 0.04) Cope: mental disengagement (0.19*; 0.03)
Ritchie T1	21.2%			18.2% AIMS2 Physical (0.44*; 0.04)	1.3% (NS)
EMS T1	11.5%				5.2% 1.4% (NS) IPQ Cause (0.25*; 0.05)

* = p<0.05, ** = p<0.01, *** p=<0.001

8.4.8 Explaining baseline health care utilization

In the cross-sectional multiple regression analysis explaining doctor visits the Kolmogorov Smirnov one sample test was significant. This indicates that the residual deviations are not normally distributed. Therefore, the data does not meet the assumptions for multiple regression and so this analysis is not reported.

8.4.9 Hypothesis 2.2 summary of findings

In the cross-sectional analyses the entire regressions were able to account for a relatively large amount of the variance in: pain (30.6-48.1%); pain (IPQ Identity and Coherence excluded; 29.2-45.1%); physical disability (70.9-71.4%); emotional distress (55.6%-66.0%) and individualised quality of life (49.4%). They explained a significant but smaller amount of variance in: social function (28.9%); occupational function (17%); disease activity (4.8-28.0%).

There is partial support for Hypothesis 2.2. Illness representations and coping procedures accounted for a significant amount of variance in health outcomes (pain, disability, emotional distress, social function, disease activity but not occupational function, individualized quality of life or health-care utilization) in the pooled sample at baseline. These results were obtained even after controlling for demographic factors, disease activity and severity, physical, emotional and social function.

More specifically, illness representations and coping accounted for significant variance in:

- | | | |
|---|--------------|----------------|
| • Pain | 3/5 analyses | (6.2 - 13.3%) |
| • Pain
(Identity & Coherence excluded) | 1/5 analyses | (10.3%) |
| • Physical disability | 2/2 analyses | (26.3 - 51.8%) |

- Emotional distress 3/3 analyses (13.1 - 29.1%)
- Social function 1/1 analysis (14.3%)
- Disease activity 3/7 analyses (4.7 - 6.6%)

Illness representations and coping procedures were found to be significant predictors of health outcomes explaining unique variance. The following associations were found (the number of analyses in which each predictor explained unique variance is given in brackets):

- Lower pain
 - lower IPQ Identity (2/5 analyses)
 - higher self-efficacy for function (3/5 analyses)
 - lower self-efficacy for other arthritis symptoms (1/5 analyses)
 - greater use of acceptance (1/5 analyses)
- Lower pain (Identity & Coherence excluded)
 - greater self-efficacy for pain (2/5 analyses)
 - greater self-efficacy for function (2/5 analyses)
 - greater use of acceptance (1/5 analyses)
- Lower physical disability
 - greater self-efficacy for function (2/2 analyses)
 - lower self-efficacy for other arthritis symptoms (1/2 analyses)
 - less use of behavioural disengagement (1/2 analyses)
 - less use of focusing on/venting emotions (1/2 analyses)

- **Lower emotional distress**
 - lower IPQ Identity (2/3 analyses)
 - perceiving RA as less puzzling/distressing (1/3 analyses)
 - perceiving the consequences of RA as less serious (1/3 analyses)
 - perceiving RA as more controllable/curable (1/3 analyses)
 - less use of suppression of competing activities (1/3 analyses)
 - less use of behavioural disengagement (3/3 analyses)
 - less use of focusing on/venting emotions (2/3 analyses)
 - more use of acceptance (1/3 analyses)
- **Higher social function**
 - higher IPQ Identity (1/1 analyses)
 - higher self-efficacy for other arthritis symptoms (1/1 analyses)
 - more use of seeking emotional social support (1/1 analyses)
- **Greater individualized quality of life**
 - perceiving the consequences of RA as less serious (1/1 analyses)
- **Lower disease activity**
 - making fewer causal attributions for RA (4/7 analyses)
 - less use of mental disengagement (1/7 analyses)

Emotional variables also accounted for a significant amount of variance in health outcomes. Together emotional state, illness representations (including IPQ Identity and Coherence) and coping procedures accounted for more variance than socio-demographic, disease activity, pain, physical and social function variables in 10 of 20 analyses. In only one analysis was an immunological measure, ESR, found to be a significant predictor of a health outcome, anxiety.

8.5 Do baseline illness representations and coping procedures predict future health outcomes?

The results of the multiple regression analyses computed to examine predictors of health outcomes at times 3, 4 and 5 are described in Appendix K. For the sake of parsimony, usually only the final models are described. However, when model 5 explains significant additional variance, the full results of this model are also presented. This allows examination of how baseline illness representation and coping procedure predictors change when the concurrent illness representations and coping variables are entered in the final model.

The results are also summarized in Table form in Appendix K (Tables K.1-8). The tables include the total amount of variance in the dependent variable explained by the final model (Adjusted R^2 % variance) and the additional variance explained at each significant step in the analysis (Adjusted R^2 % change; $\Delta adj.R^2$). The tables also include the significant predictors, from the final model, presented under the column representing the step at which they were first entered in the analysis. When baseline illness representations and coping procedures, entered in model 5, explained significant additional variance the significant predictors are also included in the table. For each significant predictor the beta weight (β), and unique variance explained (r_{sp}^2) are also presented in the table and in the text. The specific findings related to Hypothesis 2.3 are described below and those for Hypothesis 2.4 are summarized in section 8.6.

Hypothesis 2.3 Baseline illness representations and coping procedures will predict a significant amount of variance in the following health outcomes in the pooled sample (independent of that accounted for by experimental condition) at time points 3-5:

- a. Pain
- b. Physical disability
- c. Emotional distress
- d. Social/Occupational function

- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

This hypothesis explores Leventhal's self-regulatory model which proposes that illness representations shape coping procedures which in turn will influence future health outcomes. Baseline illness representation, coping, self-management and self-efficacy measures were found to predict the following outcome measures later in the study.

8.5.1 Predicting pain time 3, 4 and 5

The results of the multiple regression analyses predicting pain are shown in Table K.1 in Appendix K.

NRIP

When entered in model 5, baseline illness representations and coping procedures did not explain a significant amount of additional variance in pain (NRIP), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance.

At time 3, lower pain was predicted by higher baseline IPQ Identity (-0.37*; 0.03) and lower baseline self-efficacy for pain (0.28*; 0.03).

At time 4, Lower pain was predicted by lower baseline self-efficacy for pain (0.26*: 0.02)

At time 5, lower pain was predicted by higher baseline IPQ Identity (-0.31*; 0.03) lower baseline self-efficacy for pain (0.30*: 0.04)

GRSP

When entered in model 5, baseline illness representations and coping procedures did not explain a significant amount of additional variance in pain (GRSP), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance.

At time 3, lower pain was predicted by lower baseline self-efficacy for pain (0.25*; 0.02), higher use of restraint coping as a coping strategy at baseline (-0.21*: 0.03).

At time 4, lower pain was predicted by lower baseline self-efficacy for pain (0.30*; 0.04).

At time 5, lower pain was predicted by lower baseline self-efficacy for pain (0.21*; 0.02).

BVAS

When entered in model 5, baseline illness representations and coping procedures did not explain a significant amount of additional variance in pain (BVAS), at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Lower pain was predicted by lower baseline self-efficacy for pain (0.25*; 0.02), less use of denial as a coping strategy at baseline (0.18*; 0.02), greater use of restraint coping as a coping strategy at baseline (-0.19*; 0.02).

Pain distress

When entered in model 5, baseline illness representations and coping procedures did not explain a significant amount of additional variance in pain distress, at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Lower pain distress was predicted by lower baseline self-efficacy for pain (0.34**; 0.04).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 7.8% variance in pain distress at time 4. In model 5, lower pain distress was predicted by perceiving RA symptoms as less puzzling/distressing (0.22*; 0.03) and greater practice of stretching exercises (-0.28**; 0.06), at baseline. In the final model, lower pain distress was predicted by higher baseline practice of stretching exercises (-0.18*; 0.03).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 11.4% variance in pain distress at time 5. In model 5, lower pain distress was predicted by perceiving the consequences of RA as more serious (-0.24*; 0.03), perceiving RA symptoms as less puzzling/distressing (0.32**; 0.06) and greater practice of stretching exercises (-0.26**; 0.05) at baseline. In the final model, lower pain distress was predicted by greater practice of stretching exercises at baseline (-0.17*; 0.02), and greater use of ignoring as a pain coping strategy at baseline (-0.20*; 0.02).

AIMS2 Symptoms

When entered in model 5, baseline illness representations and coping procedures did not explain a significant amount of additional variance in pain (AIMS2 Symptoms), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance at times 4 and 5.

At time 4, lower pain was predicted by lower baseline self-efficacy for pain (0.27*; 0.02).

At time 5, lower pain was predicted by lower baseline self-efficacy for pain (0.32**; 0.04).

8.5.2 Predicting pain time 3, 4 and 5 (excluding IPQ Identity and IPQ Coherence)

The results of the multiple regression analyses predicting pain (excluding IPQ Identity from all analyses and IPQ Coherence from analyses where pain distress was the dependent variable) are presented in Table K.2.

NRIP

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in pain (NRIP), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance at times 3 and 5.

At time 3, Lower pain was predicted by lower baseline self-efficacy for pain (0.27*; 0.03).

At time 5, lower pain was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.24*; 0.03), lower baseline self-efficacy for pain (0.30*; 0.03).

GRSP

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in pain (GRSP), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance at times 3 and 5.

At time 3, lower pain was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.25*; 0.02), lower baseline self-efficacy for pain (0.25*; 0.02), greater use of restraint coping as a coping strategy at baseline (-0.24**; 0.04).

At time 5, lower pain was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.35**; 0.04).

BVAS

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in pain (BVAS), at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Lower pain was predicted by lower baseline self-efficacy for pain (0.26*; 0.03).

Pain distress

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in pain distress, at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance.

At time 3, lower pain distress was predicted by lower baseline self-efficacy for pain (0.33*; 0.04), lower baseline generalised self-efficacy (0.30*; 0.03).

At time 4, lower pain distress was predicted by greater use of stretching exercises at baseline (-0.21*; 0.04).

At time 5, lower pain distress was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.29*; 0.03), lower baseline generalised self-efficacy (0.29**; 0.05), greater use of ignoring as a pain coping strategy at baseline (-0.20*; 0.02)

AIMS2 Symptoms

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in pain (AIMS2 Symptoms), at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance at times 3 and 5.

At time 3, lower pain was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.23*; 0.02).

At time 5, lower pain was predicted by perceiving RA as more serious at baseline (IPQ Consequences) (-0.27*; 0.03), lower baseline self-efficacy for pain (0.26*; 0.03).

8.5.3 Predicting physical disability time 3, 4 and 5

The results of the multiple regression analyses predicting physical disability are presented in Table K.3.

HAQ

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 2.1% variance in disability (HAQ) at time 3. In model 5, lower disability was predicted by higher self-efficacy for function at baseline (-0.25***; 0.01). In the final model, lower disability was predicted by lower baseline self-efficacy for pain (0.17**; 0.01).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 5.7% variance in disability (HAQ) at time 4. In model 5, lower disability was predicted by higher self-efficacy for function (-0.27**; 0.02), higher generalised self-efficacy (-0.18***; 0.02) and perceiving RA as more controllable/curable (-0.14**; 0.01) at baseline. In the final model, lower disability HAQ (time 4) was predicted by higher baseline generalised self-efficacy (-0.15**; 0.01), higher baseline self-efficacy for function (-0.18**; 0.00).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 2.4% variance in disability (HAQ) at time 5. In model 5, lower disability was predicted by higher self-efficacy for function at baseline (-0.30**; 0.02). In the final model, lower disability was predicted by higher baseline IPQ Identity (-0.21**; 0.01).

AIMS2 Physical

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 5.1% variance in disability (AIMS2 Physical) at time 3. In model 5, lower disability was predicted by higher self-efficacy for function at baseline (-0.37**; 0.04). In the final model, lower disability was predicted by higher baseline self-efficacy for function at baseline (-0.28**; 0.01) and perceiving RA as more controllable/curable at baseline (-0.12*; 0.01).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 7.5% variance in disability (AIMS2 Physical) at time 4. In model 5, lower disability was predicted by perceiving RA symptoms as less puzzling/distressing (0.21*; 0.03), higher self-efficacy for function (-0.26*; 0.02), higher generalised self-efficacy (-0.16**; 0.01), greater use of seeking instrumental social support (-0.15**; 0.02) and less use of ignoring as a pain coping strategy (0.17**; 0.01) at baseline. In the final model, lower disability was predicted by perceiving RA symptoms as less puzzling/distressing at baseline (IPQ Coherence) (0.16*; 0.01), greater generalised self-efficacy at baseline (-0.16**; 0.01), greater use of seeking instrumental social support as a coping strategy at baseline (-0.13**; 0.01).

8.5.4 Predicting emotional distress time 3, 4 and 5

The results of the multiple regression analyses predicting emotional distress are presented in Table K.4.

HADS depression

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in depression (HADS) at time 5. However, in the final model they did emerge as significant predictors explaining unique variance. Lower

depression HADS was predicted by less use of restraint coping as a coping strategy at baseline (0.15*; 0.02).

HADS Anxiety

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in anxiety (HADS) at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance.

At time 3, lower anxiety HADS was predicted by, lower baseline self-efficacy for other arthritis symptoms (0.25*; 0.02).

At time 4, lower anxiety HADS was predicted by less use of diverting attention as a pain coping strategy at baseline (0.15*; 0.02), greater use of focusing on and venting emotions as a coping strategy at baseline (-0.21*; 0.02), greater use of denial as a coping strategy at baseline (-0.17*; 0.02).

At time 5, lower anxiety was predicted by greater use of denial as a coping strategy at baseline (-0.18*; 0.02).

AIMS2 Affect

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 4.5% variance in emotional distress (AIMS2 Affect) at time 4. In both model 5 and 6, lower emotional distress was predicted by higher baseline self-efficacy for pain (-0.23*; 0.03).

8.5.5 Predicting social and occupational function time 3, 4 and 5

The results of the multiple regression analyses predicting social and occupational function are presented in Table K.5.

AIMS2 Social

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in social function at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Greater social function was predicted by lower baseline self-efficacy for other arthritis symptoms (0.25*; 0.02).

8.5.6 Predicting individualized quality of life time 3, 4 and 5

The results of the multiple regression analyses predicting individualized quality of life are presented in Table K.6.

PGI

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in individualized quality of life PGI at time 3, 4 or 5. However, in the final model they did emerge as significant predictors explaining unique variance at times 3 and 4.

At time 3, better individualized quality of life was predicted by greater IPQ Identity at baseline (0.32*; 0.02), less use of restraint coping as a coping strategy at baseline (-0.22**; 0.03).

At time 4, better individualized quality of life was predicted by less use of restraint coping as a coping strategy at baseline (-0.21*: 0.02), greater use of focusing on and venting emotions as a coping strategy at baseline (0.24*; 0.02).

8.5.7 Predicting disease activity time 3, 4 and 5

The results of the multiple regression analyses predicting disease activity are presented in Table K.7.

CRP

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 17.8% variance in disease activity (CRP) at time 3. In model 5, the final model, lower disease activity was predicted by higher self-efficacy for function (-0.31**; 0.03), less practice of relaxation (0.39***; 0.14) at baseline.

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 9.2% variance in disease activity (CRP) at time 4. In model 5, lower disease activity was predicted by less practice of relaxation (0.24***; 0.05) and less use of seeking instrumental social support (0.17**; 0.02) at baseline. In model 6, the final model, lower disease activity was predicted by greater self-efficacy for function at baseline (-0.33*; 0.03), less practice of relaxation at baseline (0.24*; 0.04), less use of seeking instrumental social support as a coping strategy at baseline (0.20; 0.03).

Ritchie Index

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 3.0% variance in tender joint score at time 3. In model 5, the final model, lower tender joint score was predicted by perceiving RA to be less chronic, IPQ timeline (0.12*; 0.01) and less practice of relaxation (0.13*; 0.01) at baseline.

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in tender joint score at time 4. However, in the

final model they did emerge as significant predictors explaining unique variance. Lower tender joint score was predicted by higher self-efficacy for function at baseline (-0.28; 0.02).

EMS

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 3.7% variance in early morning stiffness at time 4. In model 5, shorter duration of early morning stiffness was predicted by less practice of stretching exercises at baseline (0.24**; 0.05). In the final model, shorter duration of early morning stiffness was predicted by less practice of stretching exercises at baseline (0.28***; 0.07).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 6.5% variance in early morning stiffness at time 5. However, in models 5 and 6 there were no specific significant illness representations or coping procedures variables explaining unique variance.

HB

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 3.0% variance in haemoglobin at time 5. In model 5, higher haemoglobin was predicted by higher generalised self-efficacy (0.16**; 0.02) less use of restraint coping as a coping strategy (-0.14*; 0.02) at baseline. In model 6, higher haemoglobin was predicted by perceiving the consequences of RA as more serious (IPQ Consequences) at baseline (0.17**; 0.02).

WCC

When entered in model 5, baseline illness representations and coping procedures did not explained significant additional variance in lower white cell count at time 4. However, in

the final model they did emerge as significant predictors explaining unique variance. Lower white cell count was predicted by lower self-efficacy for pain at baseline (0.21**; 0.02).

Platelets

When entered in model 5, baseline illness representations and coping procedures did not explain significant additional variance in platelets at time 5. However, in the final model they did emerge as significant predictors explaining unique variance. Lower platelets was predicted by less use of positive interpretation/growth as a coping strategy at baseline (0.12*; 0.01).

8.5.8 Predicting health-care utilization time 3, 4 and 5

The results of the multiple regression analyses predicting doctor visits are presented in Table K.8.

Doctor Visits

When entered in model 5, baseline illness representations and coping procedures did not explained significant additional variance in frequency of doctor visits at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Lower frequency of doctor visits was predicted by less use of seeking instrumental social support as a coping strategy at baseline (0.22*; 0.04).

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 10.9% variance in frequency of doctor visits at time 4. In model 5, the final model, lower frequency of doctor visits was predicted by greater use of restraint coping as a coping strategy at baseline (-0.31***; 0.08)

When entered in model 5, baseline illness representations and coping procedures explained a significant additional 5.5% variance in frequency of doctor visits at time 5.

- Emotional distress 1/9 analyses (4.5%)
 - AIMS2 Affect 4 (4.5%)

- Disease activity 6/17 analyses range = (3.0-17.8%)
 - mean = 7.2%
 - CRP 3 (17.8%)
 - CRP 4 (9.2%)
 - Ritchie 3 (3.0%)
 - EMS 4 (3.7%)
 - EMS 5 (6.5%)
 - HB 5 (3.0%)

- Health-care utilization 2/3 analyses range = (5.5-10.9%)
 - mean = 8.2%
 - Doctor Visits 4 (10.9%)
 - Doctor Visits 5 (5.5%)

Overall, the amount of variance explained was modest, usually less than 10% (range=2.1-17.8%) and was only found in 16/74 analyses. However, these results were found after controlling for the baseline of the dependent variable, socio-demographic factors, disease activity, physical, social and emotional function, and up to a year later.

When entered in model 5 specific Baseline illness representations and coping procedures were found to be significant predictors explaining unique variance in health outcomes. The following predictive relationships were found in model 5:

- Lower pain distress
 - perceiving RA as having more serious consequences (1/2 analyses)
 - perceiving RA as less puzzling/distressing (2/2 analyses)
 - greater practice of stretching exercises (2/2 analyses)

The most consistent predictors of lower pain distress were perceiving RA as less puzzling/distressing (2/2 analyses) and greater practice of stretching exercises (2/2 analyses).

- Lower physical disability
 - perceiving RA symptoms as less puzzling/distressing (1/5 analyses)
 - perceiving RA as more controllable (1/5 analyses)
 - higher self-efficacy for functional activities (5/5 analyses)
 - higher generalized self-efficacy (2/5 analyses)
 - greater use of seeking instrumental social support (1/5 analyses)
 - less use of ignoring pain sensations (1/5 analyses)

The most consistent predictors of lower physical disability were higher self-efficacy for functional activities (5/5 analyses) and higher generalized self-efficacy (2/5 analyses).

- Lower emotional distress
 - higher self-efficacy for pain (1/1 analyses)
- Lower disease activity
 - perceiving RA as less chronic (IPQ Timeline) (1/6 analyses)
 - higher self-efficacy for functional activities (1/5 analyses)
 - higher generalized self-efficacy (1/5 analyses)
 - less practice of relaxation (3/5 analyses)
 - less practice of stretching exercises (1/5 analyses)
 - less use of seeking instrumental social support (1/5 analyses)
 - less use of restraint coping (1/5 analyses)

The most consistent predictor of lower disease activity was less practice of relaxation (3/5 analyses).

- Lower frequency doctor visits
 - greater use restraint coping (1/2 analyses)
 - less use of positive interpretation/growth (1/2 analyses)

Baseline illness representations and coping procedures frequently did not explain a significant amount of additional variance when entered in model 5. However, when concurrent illness representation and coping variables were entered in model 6 new baseline illness representation and coping variables frequently emerged as significant predictors. The direction of the association between the predictor, assessed at baseline, and the outcome measure was frequently opposite to that between the same predictor, assessed concurrently, and the health outcome. This reflects the interaction between baseline and concurrent illness representation and coping variables. More maladaptive scores at baseline provide more opportunity for change to a more adaptive concurrent score. The following predictive relationships were found between baseline illness representations and coping procedures and health outcomes in model 6:

- Lower pain
 - higher IPQ Identity (2/15 analyses)
 - lower self-efficacy for pain (10/15 analyses)
 - greater use of restraint coping (2/15 analyses)
 - greater use of ignoring pain (1/15 analyses)
 - less use of denial (1/15 analyses)
 - greater practice of stretching exercises (2/15 analyses)

The most consistent predictor of lower pain was lower self-efficacy for pain (10/15 analyses).

- Lower pain (IPQ Identity & Coherence excluded)
 - greater perceived consequences (IPQ Consequences) (6/15 analyses)
 - lower self-efficacy for pain (6/15 analyses)
 - lower generalized self-efficacy (2/15 analyses)

greater use of restraint coping	(1/15 analyses)
greater use of ignoring pain	(1/15 analyses)
greater practice of stretching exercises	(1/15 analyses)

The most consistent predictors of lower pain (when IPQ Identity and Coherence were excluded) were perceiving the consequences of RA as more serious (6/15 analyses) and lower self-efficacy for pain (6/15 analyses).

- Lower physical disability

higher IPQ Identity	(1/6 analyses)
perceiving RA symptoms as less puzzling and distressing (IPQ Coherence)	(1/6 analyses)
perceiving RA as more controllable (IPQ Control/Cure)	(1/6 analyses)
lower self-efficacy for pain	(1/6 analyses)
higher self-efficacy for function	(2/6 analyses)
higher generalized self-efficacy	(2/6 analyses)
greater use of seeking instrumental social support	(1/6 analyses)

The most consistent predictors of lower physical disability were greater self-efficacy for function (2/6 analyses) and greater generalized self-efficacy (2/6 analyses).

- Lower emotional distress

higher self-efficacy for pain	(1/9 analyses)
lower self-efficacy for other arthritis symptoms	(1/9 analyses)
greater use of focusing/venting emotions	(1/9 analyses)
greater use of denial	(2/9 analyses)
less use of restraint coping	(1/9 analyses)
less use of diverting attention	(1/9 analyses)

The most consistent predictor of lower emotional distress was greater use of denial as a coping strategy (2/9 analyses).

- **Higher social function**
 lower self-efficacy for other arthritis symptoms (1/3 analyses)
- **Greater individualized quality of life**
 greater IPQ Identity (1/3 analyses)
 greater use of restraint coping (2/3 analyses)
 greater use of focusing/venting emotions (1/3 analyses)

The most consistent predictor of higher quality of life was greater use of restraint coping (2/3 analyses).

- **Lower disease activity**
 perceiving RA as less chronic (IPQ Timeline) (1/17 analyses)
 perceiving the consequences of RA as less serious (1/17 analyses)
 higher self-efficacy for function (3/17 analyses)
 lower self-efficacy for pain (1/17 analyses)
 less use of positive interpretation/growth (1/17 analyses)
 less practice of relaxation exercises (3/17 analyses)
 less practice of stretching exercises (1/17 analyses)
 less use of seeking instrumental social support (1/17 analyses)

The most consistent predictors of lower disease activity were higher self-efficacy for function (3/17 analyses) and less practice of relaxation exercises (3/17 analyses).

- **Lower number of doctor visits**
 greater use of restraint coping (1/3 analyses)
 less use of seeking instrumental social support (1/3 analyses)
 less use of positive interpretation/growth (1/3 analyses)

8.6 Do changes in illness representations and coping procedures predict future health outcomes?

The complete results of the multiple regression analyses computed to examine predictors of health outcomes at time 3, 4 and 5 are described in Appendix K. The results are summarized in Tables K.1-8. The tables include the total amount of variance in the dependent variable explained by the final model (Adjusted R^2 % variance) and the additional variance explained at each significant step in the analysis (Adjusted R^2 % change; $\Delta adj.R^2$). The tables also include the significant predictors, from the final model, presented under the column representing the step at which they were first entered in the analysis. For each significant predictor the beta weight (β), and unique variance explained (r_{sp}^2), are also presented. The specific results bearing on Hypothesis 2.4 are summarized below.

Hypothesis 2.4 Change in illness representations and coping procedures will explain a significant amount of variance in health outcomes in the pooled sample at time points 3-5:

- a. Pain
- b. Physical disability
- c. Emotional distress
- d. Social/Occupational Function
- e. Quality of life (PGI)
- f. Disease activity
- g. Health Care utilization

Leventhal's self regulatory model proposes that changes in illness representations and coping procedures should influence health outcomes through dynamic self regulatory processes. This hypothesis tests the extent to which changes in illness representations and coping procedures contribute to change in RA health outcomes. Since there are statistical problems using change scores, the method of analysis chosen was to use the predictor variable at the same time point as the criterion variable but control for baseline

scores of both variables. Controlling for baseline levels, concurrent illness representations (illness perceptions and self-efficacy) and coping procedures (coping strategies and self management behaviours) accounted for a significant amount of variance in the following health outcomes at 3, 6 and 12 months.

8.6.1 Predicting pain time 3, 4 and 5

The results of the multiple regression analyses predicting pain are presented in Table K.1.

NRIP

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 16.8% variance in pain (NRIP) at time 3. Lower pain was predicted by lower IPQ Identity (0.53***; 0.08).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 20.0% variance in pain (NRIP) at time 4. Lower pain was predicted by lower IPQ Identity (0.58***; 0.10) and higher self-efficacy for pain (-0.3*; 0.02).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 24.4% variance in pain (NRIP) at time 5. Lower pain was predicted by lower IPQ Identity (0.47***; 0.08).

GRSP

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 15.7% variance in pain (GRSP) at time 3. Lower pain was predicted by lower IPQ Identity (0.46***; 0.06), perceiving the consequences of RA as less serious (IPQ Consequences; 0.23*; 0.02) and less use of restraint coping as a coping strategy (0.21*; 0.03).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 12.8% variance in pain (GRSP) at time 4. Lower pain was predicted by lower IPQ Identity (0.52***; 0.08), higher self-efficacy for pain (-0.29*; 0.03) and less use of restraint coping as a coping strategy (0.22*; 0.04).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 20.7% variance in pain (GRSP) at time 5. Lower pain was predicted by lower IPQ Identity (0.49***; 0.08).

BVAS

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 19.9% variance in pain (BVAS) at time 3. Lower pain was predicted by lower IPQ Identity (0.48***; 0.06), perceiving symptoms of RA as less puzzling/distressing (IPQ Coherence; 0.26*; 0.03) and greater self-efficacy for other arthritis symptoms (-0.34*; 0.03).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 15.0% variance in pain (BVAS) at time 4. Lower pain was predicted by lower IPQ Identity (0.50***; 0.07).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 11.7% variance in pain (BVAS) at time 5. Lower pain was predicted by lower IPQ Identity (0.35*; 0.04).

Pain Distress

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 18.3% variance in pain distress at time 3. Lower pain

distress was predicted by lower IPQ Identity (0.46**; 0.05) and less use of restraint coping as a coping strategy (0.21*; 0.02).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 19.3% variance in pain distress at time 4. Lower pain distress was predicted by lower IPQ Identity (0.48***; 0.08) and perceiving RA symptoms as less puzzling/distressing (0.23*; 0.03).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 12.1% variance in pain distress at time 5. Lower pain distress was predicted by lower IPQ Identity (0.38**; 0.04).

AIMS2 Symptoms

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 14.6% variance in pain (AIMS2 Symptoms) at time 3. Lower pain was predicted by lower IPQ Identity (0.48***; 0.04), higher self-efficacy for other arthritis symptoms (-0.25*; 0.01) and greater use of reinterpretation as a pain coping strategy (-0.25***; 0.03).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 14.9% variance in pain (AIMS2 Symptoms) at time 4. Lower pain was predicted by lower IPQ Identity (0.45***; 0.05) and higher self-efficacy for pain (-0.28*; 0.02).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 24.5% variance in pain (AIMS2 Symptoms) at time 5. Lower pain was predicted by lower IPQ Identity (0.57***; 0.10), higher self-efficacy for pain (-0.41*; 0.10).

8.6.2 Predicting pain time 3, 4 and 5 (excluding IPQ Identity and IPQ Coherence)

The results of the multiple regression analyses predicting pain (excluding IPQ Identity from all analyses and IPQ Coherence from analyses where pain distress was the dependent variable) are presented in Table K.2.

NRIP

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 7.5% variance in pain (NRIP) at time 4. Lower pain was predicted by higher self-efficacy for pain (-.033*; 0.03)

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 15.4% variance in pain (NRIP) at time 5. Lower pain was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.28*; 0.04) and higher Self Efficacy for pain (-0.46**; 0.06).

GRSP

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 10.2% variance in pain (GRSP) at time 3. Lower pain was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.29*; 0.03) and less use of restraint coping as a coping strategy (0.19*; 0.02).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 13.0% variance in pain (GRSP) at time 4. Lower pain was predicted by higher self-efficacy for pain (-0.35**; 0.05) and less use of restraint coping as a coping strategy (0.23*; 0.04).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 11.1% variance in pain (GRSP) at time 5. Lower pain was predicted by perceiving the symptoms of RA as less puzzling/distressing (IPQ Coherence; 0.21*; 0.02), greater self-efficacy for pain (-0.29*; 0.03) and greater self-efficacy for function (-0.35**; 0.02).

BVAS

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 14.3% variance in pain (BVAS) at time 3. Lower pain was predicted by perceiving the symptoms of RA as less puzzling/distressing (IPQ Coherence; 0.22*; 0.02) and greater self-efficacy for other arthritis symptoms (-0.38*; 0.04)

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 7.4% variance in pain (BVAS) at time 5. Lower pain was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.25*; 0.03).

Pain Distress

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 11.3% variance in pain distress at time 3. Lower pain distress was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.28*; 0.03) and higher self-efficacy for other arthritis symptoms (-0.34*; 0.03).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 8.1% variance in pain distress at time 4. Lower pain distress was predicted by less use of increased behaviour as a pain coping strategy (0.30*; 0.04).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 7.8% variance in pain distress at time 5. Lower pain distress was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.23*; 0.02).

AIMS2 Symptoms

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 9.0% variance in pain (AIMS2 Symptoms) at time 3. Lower pain was predicted by greater use of reinterpretation as a pain coping strategy (-0.22*; 0.02).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 7.2% variance in pain (AIMS2 Symptoms) at time 4. Lower pain was predicted by higher self-efficacy for pain (-0.33*; 0.03).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 12.4% variance in pain (AIMS2 Symptoms) at time 5. Lower pain was predicted by greater self-efficacy for pain (-0.5***; 0.07).

8.6.3 Predicting physical disability time 3, 4 and 5

The results of the multiple regression analyses predicting physical disability are presented in Table K.3.

HAQ

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 3.9% variance in disability (HAQ) at time 3. Lower disability was predicted by lower IPQ Identity (0.22**; 0.01), perceiving the

consequences of RA as less serious (IPQ Consequences; 0.13*; 0.01) and greater self-efficacy for pain (-0.18*; 0.01).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 5.0% variance in disability (HAQ) at time 4. Lower disability was predicted by lower IPQ Identity (0.18**; 0.01), greater self-efficacy for function (-0.19**; 0.00) and higher frequency of walking (-0.18**; 0.01).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 6.5% variance in disability (HAQ) at time 5. Lower disability was predicted by lower IPQ Identity (0.25***; 0.02) and greater self-efficacy for function (-0.28*; 0.01).

AIMS2 Physical

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 4.2% variance in disability (AIMS2 Physical) at time 4. Lower disability was predicted by greater self-efficacy for function (-0.21*; 0.01).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 7.2% variance in disability (AIMS2 Physical) at time 5. Lower disability was predicted by greater self-efficacy for pain (-0.19*; 0.00) and greater self-efficacy for function (-0.27*; 0.01).

8.6.4 Predicting emotional distress time 3, 4 and 5

The results of the multiple regression analyses predicting emotional distress are presented in Table K.4.

HADS Depression

When entered in model 6, illness representations and coping procedures at time 3 did not explain significant additional variance in depression (HADS) at time 3. However, in the final model they did emerge as significant predictors explaining unique variance. Lower depression was predicted by higher self-efficacy for other arthritis symptoms (-0.33*; 0.02).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 10.3% variance in depression (HADS) at time 4. Lower depression was predicted by less use of focusing on and venting emotions as a coping strategy (0.22**; 0.03).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 3.9% variance in depression (HADS) at time 5. However, no specific illness representation or coping procedure predictors explained significant unique variance.

HADS Anxiety

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 16.6% variance in anxiety (HADS) at time 3. Lower anxiety was predicted by lower IPQ Identity (0.30**; 0.02), greater self-efficacy for other arthritis symptoms (-0.28**; 0.02) and less use of focusing on and venting emotions as a coping strategy (0.17*; 0.02).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 9.3% variance in anxiety (HADS) at time 4. Lower anxiety was predicted by lower IPQ Identity (0.29*; 0.02), less use of focusing on and venting emotions as a coping strategy (0.26***; 0.04) and less use of denial as a coping strategy (0.17*; 0.02).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 7.2% variance in anxiety (HADS) at time 5. Lower anxiety was predicted by lower IPQ Identity (0.34**; 0.04).

AIMS2 Affect

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 14.5% variance in emotional distress (AIMS2 Affect) at time 3. Lower emotional distress was predicted by higher self-efficacy for other arthritis symptoms (-0.55***; 0.07).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 17.3% variance in emotional distress (AIMS2 Affect) at time 5. Lower emotional distress was predicted by higher self-efficacy for other arthritis symptoms (-0.31*; 0.02) and less use of focusing on and venting emotions as a coping strategy (0.29**; 0.04).

8.6.5 Predicting social/occupational function time 3, 4 and 5

The results of the multiple regression analyses predicting social and occupational function are presented in Table K.5.

AIMS2 Social

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 6.1% variance in social function (AIMS2 Social) at time 3. Greater social function was predicted by greater self-efficacy for other arthritis symptoms (-0.29*; 0.02) and greater use of increased behaviour as a coping strategy (-0.32*; 0.02).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 11.0% variance in social function (AIMS2 Social) at time 5. Greater social function was predicted by greater use of increased behaviour as a coping strategy (-0.29*; 0.02) and greater use of seeking emotional social support as a coping strategy (-0.34***; 0.06).

AIMS2 Role

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 5.7% variance in occupational function (AIMS2 Role) at time 3. Greater occupational function was predicted by greater use of seeking instrumental social support as a coping strategy (-0.23*; 0.03) and perceiving the consequences of RA as less serious (IPQ Consequences; 0.26*; 0.03).

8.6.6 Predicting individualized quality of life time 3, 4 and 5

The results of the multiple regression analyses predicting individualized quality of life are presented in Table K.6.

PGI

When entered in model 6, illness representations and coping procedures at time 3 explained a significant additional 12.4% variance in individualized quality of life PGI at time 3. Better individualized quality of life was predicted by lower IPQ Identity (-0.45**; 0.05), greater generalised self-efficacy (0.26*; 0.02) and greater use of ignoring pain as a pain coping strategy (0.34*; 0.03).

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 7.5% variance in individualized quality of life PGI at time 4. Better individualized quality of life was predicted by lower IPQ Identity (-0.29*; 0.02), and perceiving RA as less chronic (IPQ timeline; (-0.30**; 0.04).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 13.3% variance in individualized quality of life PGI at time 5. Better individualized quality of life was predicted by perceiving the consequences of RA as less serious (-0.27*; 0.02).

8.6.7 Predicting disease activity time 3, 4 and 5

The results of the multiple regression analyses predicting disease activity are presented in Table K.7.

EMS

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 4.7% variance in early morning stiffness at time 4. Shorter duration of early morning stiffness was predicted by perceiving the consequences of RA as less serious (IPQ Consequences; 0.24*; 0.03).

When entered in model 6, illness representations and coping procedures at time 5 explained a significant additional 7.3% variance in early morning stiffness at time 5. Shorter duration of early morning stiffness was predicted by perceiving the consequences of RA as less serious IPQ Consequences; 0.30*; 0.04).

WCC

When entered in model 6, illness representations and coping procedures at time 4 explained a significant additional 2.6% variance in white cell count at time 4. Lower white cell count was predicted by perceiving RA to be less chronic (IPQ Timeline; 0.15*; 0.01).

Platelets

When entered in model 6, illness representations and coping procedures at time 5 did not explain significant additional variance in platelets at time 5. However, in the final model they did emerge as significant predictors explaining unique variance. Lower platelets was predicted by greater frequency of walking at time 5 (-0.16*; 0.01).

8.6.8 Hypothesis 2.4 summary of findings

In summary there is partial support for Hypothesis 2.4. Controlling for baseline scores, concurrent illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social function, occupational function, quality of life and disease activity but not health-care utilization) in the pooled sample at time points 3, 4 or 5.

More specifically, concurrent illness representations and coping accounted for significant variance in the following health outcomes at time 3, 4 or 5:

- Pain 15/15 analyses range = (11.7-24.5%)
mean = 17.4%
- Pain 15/15 analyses range = (7.4-15.4%)
(IPQ Identity and Coherence excluded) mean = 10.0%
- Physical disability 5/6 analyses range = (1.9-7.2%)
mean = 4.8%
- Emotional distress 7/9 analyses range = (3.9-17.3%)
mean = 11.3%

- **Social Function** **2/3 analyses** **range = (6.1-11.0%)**
mean = 8.6%
- **Occupational Function** **1/3 analyses** **range = (5.7%)**
mean = 5.7%
- **Quality of life** **3/3 analyses** **range = (7.5-13.3%)**
mean = 11.1%
- **Disease activity** **3/17 analyses** **range = 2.6-7.3%**
Mean = 4.9%

Overall, concurrent illness representations and coping procedures explained a small to moderate amount of variance (range=1.9-24.5%). The results were consistent being found in 51 of 74 analyses. Illness representations and coping procedures most consistently predicted pain, disability, emotional distress, social function and quality of life. The results are impressive as they were found after controlling for the baseline of the dependent variable, socio-demographic factors, disease activity, physical, social and emotional function, and baseline illness representations and coping procedures. Apart from the baseline of the dependent variable, concurrent illness representations and coping procedures were the most important predictors of health outcomes.

In the final model, concurrent illness representations and coping procedures frequently emerged as significant predictors explaining unique variance in health outcomes. The following predictive relationships were found:

- **Lower pain**
 - lower IPQ Identity (15/15 analyses)
 - less puzzling/distressing (IPQ Coherence) (2/15 analyses)
 - less serious IPQ Consequences (1/15 analyses)
 - higher self-efficacy for pain (4/15 analyses)

higher self-efficacy for other arthritis symptoms	(2/15 analyses)
less use of restraint coping	(3/15 analyses)
greater use of reinterpreting pain sensations	(1/15 analyses)

The most consistent predictors of lower pain were lower IPQ Identity (15/15 analyses), greater self-efficacy for pain (4/15 analyses) and less use of restraint coping (3/15 analyses).

- Lower pain (IPQ Identity and Coherence excluded)

less serious (IPQ Consequences)	(5/15 analyses)
less puzzling/distressing (IPQ Coherence)	(2/15 analyses)
higher self-efficacy for pain	(6/15 analyses)
higher self-efficacy for function	(1/15 analyses)
higher self-efficacy for other arthritis symptoms	(2/15 analyses)
less use of restraint coping	(2/15 analyses)
less use of increased behaviour	(1/15 analyses)
greater use of reinterpreting pain sensations	(1/15 analyses)

When IPQ Identity and Coherence were excluded from the analyses, the most consistent predictors of lower pain were lower perceived Consequences (5/15 analyses), and greater self-efficacy for pain (6/15 analyses).

- Lower physical disability

lower IPQ Identity	(3/6 analyses)
less serious (IPQ Consequences)	(1/6 analyses)
higher self-efficacy for pain	(2/6 analyses)
higher self-efficacy for function	(4/6 analyses)
higher frequency of walking	(1/6 analyses)

The most consistent predictors of lower disability were lower IPQ Identity (3/6 analyses), greater self-efficacy for pain (2/6 analyses) and greater self-efficacy for function (4/6 analyses).

- Lower emotional distress
 - lower IPQ Identity (3/9 analyses)
 - higher self-efficacy for other arthritis symptoms (4/9 analyses)
 - less use of focusing/venting emotions (4/9 analyses)
 - less use of denial (1/9 analyses)

The most consistent predictors of lower emotional distress were lower IPQ Identity (3/9 analyses), greater self-efficacy for other arthritis symptoms (4/9 analyses) and less use of focusing/venting emotions (4/9 analyses).

- Higher social function
 - higher self-efficacy for other arthritis symptoms (1/3 analyses)
 - greater use of increased behaviour (2/3 analyses)
 - greater use of seeking emotional social support (1/3 analyses)

The most consistent predictor of greater social function was greater use of increased behaviour as a pain coping strategy (2/3 analyses).

- Higher occupational function
 - less serious (IPQ Consequences) (1/3 analyses)
 - greater use of seeking instrumental social support (1/3 analyses)

- Greater individualized quality of life**

lower IPQ Identity	(2/3 analyses)
less chronic (IPQ Timeline)	(1/3 analyses)
less serious (IPQ Consequences)	(1/3 analyses)
higher generalized self-efficacy	(1/3 analyses)
greater use of ignoring pain sensations	(1/3 analyses)

The most consistent predictor of higher individualized quality of life was lower IPQ Identity (2/3 analyses).

- Lower disease activity**

less chronic (IPQ Timeline)	(1/17 analyses)
less serious (IPQ Consequences)	(2/17 analyses)
higher frequency of walking	(1/17 analyses)

The most consistent predictor of lower disease activity was lower IPQ Consequences (2/17 analyses).

Chapter 9: The role of illness representations and coping in mediating/moderating changes in a self-management intervention

9.1 Introduction

This chapter presents the results of analyses investigating whether illness representation or coping procedures moderated (Hypothesis 3.1) or mediated (Hypothesis 3.2) the effects of the self-management intervention on health outcomes in chronic RA. The statistical procedures described by Baron and Kenny (1986) were used to test Hypothesis 3.1 and 3.2.

The analyses described in Chapter 7 identified 3 variables that changed significantly following the self-management intervention and were maintained at follow-up: individualized quality of life (PGI); self-efficacy for pain and self-efficacy for other arthritis symptoms. These variables are the focus of the analyses of mechanisms of change.

The statistical methods used to test for moderator and mediator effects are described in section 9.2. Section 9.3 presents the results of analyses examining whether illness representations and coping procedures moderate the effects of treatment (hypothesis 3.1). Section 9.4 presents the results of the analyses testing whether illness representations and coping procedures mediate the effects of treatment (Hypothesis 3.2). A summary of the findings, for both hypotheses, is presented at the end of the chapter.

9.2 Statistical methods

Testing hypotheses 3.1 and 3.2 involved performing a series of Hierarchical multiple regression analyses. For all these analyses the data was assessed to ensure it met the assumptions of linear regression, with Kolmogorov-Smirnoff one sample tests used to test the normality of the residual deviations from the regression analyses. The analysis was rejected if Z was significant at the $p=0.05$ level. In addition scatter plots were undertaken of residuals against predicted values to test the assumption of constant variance. Independent variables were usually only entered in the regression if they were significantly correlated with the dependent variable. Variables that measured the same construct as the dependent variable were not included in the regression to eliminate strong reciprocal relationships between measures.

For Hypothesis 3.1 the hierarchical multiple regression analyses were set up to test whether the criteria needed to show a moderating relationship are met (Baron & Kenny, 1986). A moderator is a variable that affects the direction and/or strength of the relationship between an independent variable (predictor) and dependent variable (criterion). The proposed moderators are continuous and the independent variable (Experimental Condition) is categorical. The purpose of the statistical procedure is to test whether the proposed moderating variable M moderates the relationship between the independent variable IV and the dependent variable DV. To test this DV is regressed on IV, M and the product of IV and M. If IV.M is significant this supports a moderation hypothesis.

In these analyses the dependent variable is the health outcome at time 3, 4 or 5. The IV, experimental condition, is entered in step 1 and the baseline of the dependent variable is entered in step 2. Possible moderators, baseline illness representation and coping procedure variables, are entered in step 3. In step 4 variables that are the product of IV and M are entered. All M and IV.M variables that are significantly correlated with DV are entered in the regression. If the possible moderator variable was not significantly correlated with the DV but the IV.M was correlated with the DV the possible moderator

is still entered in step 3 of the multiple regression in order to meet the criteria necessary to test the moderator hypothesis. Moderator relationships are indicated by a significant amount of variance being explained by Model 4.

Thus independent variables were entered in blocks in the following order:

1. Experimental condition (0 = control 1 = treatment) this was entered even if it was not significantly correlated with the dependent variable.
2. The baseline (time 1) score for the dependent variable.
3. Baseline (time 1) illness representations, coping, self management and self-efficacy variables (Time 1 scores for any variables entered in step 4 were entered even if not significantly correlated with the dependent variable to allow the testing of Hypothesis 3.1).
4. Interaction of Experimental condition and baseline illness representations, coping, self-management and self-efficacy variables (Hypothesis 3.1).

To test hypothesis 3.2 a series of hierarchical multiple regression analyses were computed to check the three criteria needed to show a mediating relationship (Baron & Kenny, 1986). A mediator accounts for the relationship between an independent variable (predictor) and dependent variable (criterion). To test for mediation a series of regression models are estimated:

1. regressing the proposed mediator (M) on the independent variable (IV).
2. regressing the dependent variable (DV) on the IV.
3. regressing the DV on both IV and M.

The variable M mediates the relationship between the IV and DV if:

1. IV accounts for variance in M
2. IV accounts for variance in DV

3. M accounts for variance in DV. The effect of the IV on the DV in the third equation must be less than in the second equation. Perfect mediation occurs if IV has no effect on DV when M is controlled.

In the first analysis the dependent variables were the outcome measures at time 3, 4 or 5. The first step (model) was the baseline of the dependent variable and the second step was experimental condition (Equation 2). Only if step 2 accounted for a significant amount of variance in the dependent variable was the next analysis computed.

In the second analysis the dependent variables were the same as in the first analysis. The independent variables were: step 1 baseline of the dependent variable and baseline of the possible mediators; step 2 the possible mediators at time 3, 4 or 5; and step 3 experimental condition (Equation 3). The third analysis was only computed if experimental condition explained significantly less variance than it did in the first analysis.

In the third analysis the dependent variable was the possible mediator (illness representation or coping variable time 3, 4 or 5) and the independent variables were: step 1; the baseline of the possible mediating variable (time 1) and step 2; experimental condition (Equation 1).

9.3 Do illness representations and coping procedures moderate the effect of a self-management intervention?

Hypothesis 3.1 Baseline illness representations and coping procedures will moderate the effect of the self-management intervention on health outcomes post treatment and/or at 3 and 9-month follow-up.

The results of the multiple regression analyses testing for moderator effects are summarized in Table 9.1. The table includes the total amount of variance in the dependent variable explained by the final model (Adjusted R^2 % variance) and the additional variance explained at each significant step in the analysis (Adjusted R^2 % change; $\Delta adj.R^2$). The tables also include the significant predictors, from the final model, presented under the column representing the step at which they were first entered in the analysis. For each significant predictor the beta weight (β), and unique variance explained (r_{sp}^2) are also presented in the table and text. Three of the analyses found significant moderator relationships, which are described below.

9.3.1 Individualized Quality Of Life

The regression explained 32.1% of the variance in individualized quality of life (PGI) at time 5. When entered in model 4, the interaction of experimental condition and illness representations and coping procedures explained a small but significant amount of variance (1.9%). In the final model, higher individualised quality of life, at the 9-month follow-up, was predicted by higher baseline quality of life (0.52***; 0.20) and in the treatment group higher baseline self-efficacy for pain (0.43; 0.03). Self-efficacy for pain moderated the effect of the intervention on PGI at time 5. People in the treatment group who had greater self-efficacy for pain at baseline were more likely to have improved quality of life at the 9-month follow-up.

9.3.3 Self-efficacy for other arthritis symptoms

The regression explained 51.1% of the variance in self-efficacy for other arthritis symptoms at time 3. When entered in model 4, the interaction of experimental condition and illness representations and coping procedures explained an additional 2.5% variance but this was not significant. However, in the final model, the interaction between experimental condition and IPQ Consequences did emerge as a significant predictor. In the final model higher self-efficacy for other arthritis symptoms at time 3 was predicted by higher baseline self-efficacy for other arthritis symptoms (0.58***; 0.20), perceiving RA as having less serious consequences at baseline (IPQ Consequences) (-0.19; 0.02) and in the treatment group perceiving RA as having more serious consequences at baseline (0.24**; 0.04). Although this does not meet the criteria for a moderating relationship, the analysis suggests that perceived Consequences may influence the effect of the intervention on self-efficacy for other arthritis symptoms at time 3. People in the treatment group who perceived RA as having more serious consequences at baseline were more likely to have improved self-efficacy for other arthritis symptoms immediately following the intervention.

The regression explained 51.3% of the variance in self-efficacy for other arthritis symptoms at time 4. When entered in model 4, the interaction of experimental condition and illness representations and coping procedures explained a significant amount of variance (8.0%). In the final model higher self-efficacy for other arthritis symptoms at time 4 was predicted by higher baseline self-efficacy for other arthritis symptoms (0.64***; 0.32), less use of ignoring pain as a coping strategy at baseline (-0.49***; 0.07) and in the treatment group greater use of ignoring pain as a coping strategy at baseline (0.90***; 0.08). Use of ignoring pain as a coping strategy at baseline moderated the effect of the intervention on self-efficacy for other arthritis symptoms at time 4. People in the treatment group who used ignoring pain as a coping strategy more at baseline were more likely to have improved self-efficacy for other arthritis symptoms at the 3-month follow-up.

The regression explained 41.9% of the variance in self-efficacy for other arthritis symptoms at time 5. When entered in model 4, the interaction of experimental condition and illness representations and coping procedures explained a small but significant amount of variance (4.6%). In the final model higher self-efficacy for other arthritis symptoms at time 5 was predicted by higher baseline self-efficacy for other arthritis symptoms (0.53***; 0.21), less use of ignoring pain as a coping strategy (-0.42**; 0.04) and in the treatment group greater use of ignoring pain as a coping strategy at baseline (0.67**; 0.04). Use of ignoring pain as a coping strategy at baseline moderated the effect of the intervention on self-efficacy for other arthritis symptoms at time 5. People in the treatment group who used ignoring pain as a coping strategy more at baseline were more likely to have improved self-efficacy for other arthritis symptoms at the 9-month follow-up.

Table 9.1 Summary of multiple regression analyses investigating moderator effects.

Dependent Variable	Adjusted R ² % Variance	INDEPENDENT VARIABLES $\Delta adj.R^2$ (β ; r_{sp}^2)			
		1 Experimental condition	2 Baseline	COGNITIONS AND COPING	
				3 Time 1	4 EC.Time 1
PGI T3	37.2%		34.1% PGI (0.38***; 0.09)		0% (NS)
PGI T4	35.3%		27.8% PGI (0.4***; 0.15)	5.6%	1.6% (NS)
PGI T5	32.1%		30.0% PGI (0.52***; 0.20)		1.9% EC Self-efficacy: Pain (0.43*; 0.03)
Self-efficacy: Pain T3	51.5%		46.1% Self-efficacy: Pain (0.64***; 0.36)		0.3 (NS)
Self-efficacy: Pain T4	46.3%		42.6% Self-efficacy: Pain (0.59***; 0.27)		0.6% (NS)
Self-efficacy: Pain T5	40.6%		40.9% Self-efficacy: Pain (0.63***; 0.38)		-0.5% (NS)
Self-efficacy: Arthritis T3	51.1%		41.8% Self-efficacy: other arthritis symptoms (0.58***; 0.20)	3.4% Consequences (-0.19*; 0.02)	2.5% (NS) EC Consequences (0.24**; 0.04)
Self-efficacy: Arthritis T4	51.3%		42.9% Self-efficacy: Arthritis (0.64***; 0.32)	Ignore (-0.49***; 0.07)	8.0% EC Ignore (0.90***; 0.08)
Self-efficacy: Arthritis T5	41.9%		32.6% Self-efficacy: Arthritis (0.53***; 0.21)	Ignore (-0.42**; 0.04)	4.6% EC Ignore (0.67**; 0.04)

* = p<0.05, ** = p<0.01, *** = p<0.001

9.4 Do illness representations and coping procedures mediate the effect of a self-management intervention?

Hypothesis 3.2 Changes in illness representations and coping procedures will mediate the effect of the self-management intervention on health outcomes post intervention and/or at 3 and 9-month follow-up.

Table 9.2-4 summarize the results of the multiple regression analyses for variables that met Baron and Kenny's (1986) criteria for identifying mediating relationships. As described in Section 9.2, to test each potential mediator three multiple regression analyses were computed (Analyses 1-3). If a second mediator was identified in the first 2 analyses then a fourth multiple regression analysis is computed for that mediator (Analysis 4). The tables include the total amount of variance in the dependent variable explained by the final model (Adjusted R² % variance) and the additional variance explained at each significant step in the analysis (Adjusted R² % change; $\Delta adj.R^2$). The tables also include the significant predictors and their beta weights (β), from each step in the analysis, presented under the column representing the step at which they were first entered. The following variables were found to mediate the effect of the intervention on individualized quality of life, self-efficacy for pain and self-efficacy for other arthritis symptoms.

9.4.1 Individualized quality of life (PGI)

IPQ Identity mediated the relationship between experimental condition and quality of life (PGI) at time 3 (see Table 9.2). Improvement in quality of life in the treatment group at time 3 was mediated by reduced IPQ Identity. People in the treatment group who reported a reduction in the perceived range and frequency of their RA symptoms were more likely to have improved quality of life following the intervention.

Table 9.2: Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on individualized quality of life at time 3.

Analysis	Dependent variable (criterion or mediator)	Adjusted R ² %	Step 1	Step 2	Step 3
		$\Delta \text{adj.}R^2$ Predictors (β)			
1	PGI T3	37.2%	35.3% PGI T1 (0.6***)	1.9% Study Group (0.16*)	
2	PGI T3	49.4%	36.3% PGI T1 (0.38***)	13.1% IPQ Identity T3 (-0.43***) Ignore T3 (0.39**)	Study Group n.s.
3	IPQ Identity T3	62%	60.6% IPQ Identity T1 (0.78***)	1.6% Study Group (-0.14*)	

9.4.2 Self-efficacy for pain

IPQ Identity mediated the relationship between experimental condition and self-efficacy for pain at time 4 (see Table 9.3). Improvement in self-efficacy for pain in the treatment group at time 4 was mediated by reduced IPQ Identity. People in the treatment group who reported a reduction in the perceived range and frequency of their RA symptoms (IPQ Identity) were more likely to have increased confidence in their ability to manage pain at the three month follow-up.

Table 9.3: Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on self-efficacy for pain at time 4.

Analysis	Dependent variable (criterion or mediator)	Adjusted R ² %	Step 1	Step 2	Step 3
		$\Delta adj.R^2$ Predictors (β)			
1	Self-efficacy: Pain T4	45.2%	42.1% Self-efficacy: Pain T1 (0.65***)	3.1% Study Group (0.19**)	
2	Self-efficacy: Pain T4	48.7%	41.3% Self-efficacy Pain T1 (0.483***)	8% IPQ Identity T4 (-0.286*)	Study Group n.s.
3	IPQ Identity T4	53.1%	51.5% IPQ Identity T1 (0.72***)	1.6% Study Group (-0.14*)	

9.4.3 Self-efficacy for other arthritis symptoms

IPQ Identity and Control Cure mediated the relationship between experimental condition and self-efficacy for other arthritis symptoms at time 3 (see Table 9.4). Improvement in self-efficacy for other arthritis symptoms in the treatment group at time 3 was mediated by reduced IPQ Identity and increased perceived controllability of RA. People in the treatment group who reported a reduction in the perceived range and frequency of their RA symptoms and increased perceived control over RA were more likely to have increased confidence in their ability to manage other RA symptoms following the intervention.

Table 9.4: Multiple regression analyses computed to identify illness representation and coping variables that may mediate the effect of self-management on self-efficacy for other arthritis symptoms at time 3.

Analysis	Dependent variable (criterion or mediator)	Adjusted R² %	Step 1	Step 2	Step 3
		Δ adj.R²: Predictors (β)			
1	Self-efficacy: Arthritis T3	45.2%	41.5% Self-efficacy: Arthritis T1 (0.65***)	3.7% Study Group (0.21**)	
2	Self-efficacy: Arthritis T3	54%	45.8% Cope: Active coping T1 (0.22**) IPQ Consequences T1 (-0.21*) Self-efficacy: Arthritis T1 (0.5***)	7.2% Relax T3 (0.23**) IPQ Identity T3 (-0.25*) IPQ Control/cure T3 (0.2*)	Study Group n.s.
3	IPQ Identity T3	62%	60.6% IPQ Identity T1 (0.78***)	1.6% Study Group (-0.14*)	
4	IPQ Control/Cure T3	26.3%	22.2% IPQ Control/cure T1 (0.48***)	4.1% Study Group (0.22**)	

9.5 Hypotheses 3.1 and 3.2 Summary of findings

9.5.1 Hypothesis 3.1: Summary of Findings

There is partial support for Hypothesis 3.1. Illness representation and coping variables moderated the effect of the intervention on some outcome measures at some time points: individualized quality of life (time 5) and self-efficacy for other arthritis symptoms (time 4 and 5).

Specifically the following moderator effects were found:

- People in the treatment group who had greater self-efficacy for pain at baseline were more likely to have improved quality of life at the 9-month follow-up.
- People in the treatment group who used ignoring pain as a coping strategy more at baseline were more likely to have improved self-efficacy for other arthritis symptoms at the 3 and 9-month follow-up.

The interaction of experimental condition and IPQ Consequences emerged as a significant predictor explaining unique variance in self-efficacy for other arthritis symptoms at time 3. People in the treatment group who perceived RA as more serious at baseline were more likely to have improved self-efficacy for other arthritis symptoms immediately following the intervention. However, the model did not explain a significant amount of additional variance so it did not meet the criteria needed for a moderator effect.

The illness representation and coping variables did not moderate the effect of the intervention on self-efficacy for pain.

Finally, the moderator effects identified were small (1.9-8.0%).

9.5.2 Hypothesis 3.2: Summary of findings

There is partial support for hypothesis 3.2. Illness representations, and coping procedures were found to mediate the relationship between experimental condition and individualized quality of life, self-efficacy for pain and self-efficacy for other arthritis symptoms at some but not all time points. Most of the mediators mediate the relationship between experimental condition and outcome variables immediately following the intervention at time 3. This suggests that changes in illness cognition and coping behaviours occurring during the intervention mediate the immediate effect of the intervention, at the time when most change occurs in the outcome measures.

The following mediator effects were identified:

- People in the treatment group who reported a reduction in the perceived range and frequency of their RA symptoms were more likely to have improved quality of life following the intervention.
- People in the treatment group who reported a reduction in the perceived range and frequency of their RA symptoms were more likely to have increased confidence in their ability to manage RA pain at the three month follow-up.
- People in the treatment group who reported a reduction in the Perceived range and frequency of their RA symptoms and increased perceived control over RA were more likely to have increased confidence in their ability to manage other RA symptoms following the intervention.

IPQ Identity mediated the effect of the intervention on several outcome measures. This suggests that a reduction in the perceived range and frequency of RA symptoms is an important mechanism in the success of the intervention.

Chapter 10: Discussion

10.1 Introduction

RA has a major impact on physical, psychological and social function but current medical management frequently fails to meet these needs (Hawley, 1995). However, given the limited efficacy of psychological interventions for chronic RA (Astin *et al.*, 2002), and methodological limitations of studies of self-management interventions, there is little high quality evidence to support the use of psycho-educational interventions for people with chronic RA. The major aim of the present dissertation was to investigate the efficacy of a self-management intervention for hospital outpatients with chronic RA in the UK.

There is considerable evidence that psychological factors are important predictors of health outcomes in chronic RA (Keefe *et al.*, 2002). The second aim of the dissertation was to explore whether Leventhal's self-regulatory model provides a useful framework for understanding the processes by which people adapt to RA. There is evidence that cognitive representations of illness and coping procedures influence health outcomes in chronic RA (Murphy *et al.*, 1999; Scharloo *et al.*, 1999). This dissertation replicates and extends this work in a longitudinal study, with a larger UK sample of people with chronic RA.

Finally, the dissertation aimed to investigate whether self-regulatory variables moderate or mediate the benefits of a self-management intervention. A better understanding of the factors predicting treatment outcome is important for a number of reasons. First, to aid identification of those people who are most/least likely to benefit from intervention. Second, to increase understanding of the mechanisms of change through which treatment is effective. Third, to give suggestions as to how interventions may be altered in order to maximise effectiveness of treatment.

10.2 The efficacy of a self-management intervention for people with chronic RA

The literature review suggested that studies of CBT for people with chronic RA have not consistently demonstrated improvements in health outcomes (Astin *et al.*, 2002; Hawley, 1995). However, the results from studies of self-management interventions have been more encouraging (Barlow *et al.*, 2000; Lorig & Holman, 1993; Taal *et al.*, 1993b). However, these studies have usually included participants recruited from community populations, with a mixture of different types of arthritis, and have significant methodological weaknesses.

This study investigated whether a self-management intervention for hospital outpatients with chronic RA produces significant changes in health outcomes (Hypothesis 1.1) and adaptive changes in illness representations and coping procedures (Hypothesis 1.2). The study tests whether findings from previous studies of self-management interventions can be generalised to a UK sample of hospital outpatients with chronic RA. The study extends previous research by using a single blind, randomised control methodology, with a baseline phase and follow-up at three and nine months.

There were four significant interaction effects on the main ANOVAs and ANCOVAs testing Hypothesis 1.1 and 1.2. The treatment group showed significantly greater improvements than the control group in individualized quality of life (PGI), self-efficacy for pain, Self-efficacy for other arthritis symptoms and use of planning as a coping strategy.

With respect to Hypothesis 1.1, the treatment group showed improvement in individualized quality of life during the baseline phase and further improvement following the intervention and at the nine-month follow-up. The analyses revealed that the treatment group improved significantly more than the control group following the intervention and this difference was maintained at the nine-month follow-up.

The treatment group's effect size, 0.45, indicated a small to moderate, but clinically significant, change in individualized quality of life between time 1 and 5. The control group's effect size, 0.08, indicates a very small and clinically insignificant change. One interpretation of this finding is that the treatment produced an improvement in quality of life. However, as some of the improvement occurred during the baseline phase this finding needs to be treated with caution.

On the main ANOVAs and ANCOVAs there was no evidence of a significant difference between the treatment and control groups over the whole study on the primary outcomes (pain, disability or emotional distress). However, for the whole sample, significant within subjects main effects on the ANOVA planned contrasts, between time 1 and 3, show significant reductions in pain (GRSP and AIMS Symptoms), disability (HAQ) and depression (HADS) following the treatment. Inspection of the graphs showed that most of the change occurred in the treatment group. This was confirmed by significant interaction effects for GRSP and HAQ and trends towards significant interaction effects for AIMS Symptoms and HADS Depression between time 1 and 3. This suggests that the treatment group showed improvements in pain, disability and depression immediately following the intervention. However, these improvements are unlikely to be fully accounted for by the intervention as some of the change occurs during the baseline phase.

It is possible that these findings might be explained by differential changes in disease activity in the treatment and control groups over the first three months of the study. However, it seems unlikely that the greater improvements in health outcomes in the treatment group could be explained by changes in disease activity as, although disease activity improved in both groups, the control group had greater improvements in both tender joint score and CRP.

It remains unclear why the treatment group showed greater improvements during the baseline phase. One possibility is a "sleeper effect". If the treatment group's disease activity had improved before entering the study this might have resulted in a subsequent improvement in health outcomes, as observed during the baseline phase. However, as

discussed in Chapter 2, disease activity is only weakly associated with pain, disability and depression. An alternative explanation is that the findings reflect an "expectancy effect". Although people did not receive the treatment during the baseline phase, the process of preparation for attending the intervention had started. People knew they would be receiving a new treatment and this may in turn have influenced their representations of the illness, coping and health outcomes. Being invited to participate in the self-management programme may have raised awareness of self-management among the treatment group participants, who may have begun to change their illness and treatment representations in anticipation of attending the ASMP. A similar effect was noted in the waiting-list control group in an RCT of the ASMP (Barlow *et al.*, 2000).

At first sight the findings are disappointing, when compared with other studies of self-management intervention which have found improvements in pain, disability and emotional state (Barlow *et al.*, 2000; Lorig & Holman, 1993; Taal *et al.*, 1993b). However, these studies did not include a baseline phase and most used a waiting list control group, which necessitated reliance on within group comparisons to assess long-term effects. The design of the Taal *et al.*, (1993b) study did allow a long-term between groups comparison. They found that the only health outcome that had improved at 4 months was disability (HAQ), and this was not maintained at 14 months. Therefore, it is not possible to be certain that the long-term improvements in health outcomes reported in most studies of the ASMP result from the intervention.

Interestingly, the results are remarkably consistent with studies of CBT for chronic RA. Very few studies of CBT have found long-term improvements in health outcomes in chronic RA, a notable exception being the study by Bradley *et al.*, (1987) which found that improvements in anxiety and depression were maintained at 6-month follow-up, but improvements in pain and disease activity found at post-treatment were not maintained at follow-up. Many studies have failed to find any significant improvements in health outcomes, pain, disability, emotional distress or joint function (Germond *et al.*, 1993; Kraimaat *et al.*, 1995c; Parker *et al.*, 1988b; Smith, Peck, Milano. & Ward, 1988). Other studies have found improvements in these health outcomes but have either not included a

long-term follow-up (Leibing *et al.*, 1999; O'Leary *et al.*, 1988; Radojevic *et al.*, 1992) or have found the improvements are not maintained at long-term follow-up (Lindroff *et al.*, 1997; Shearn & Fireman, 1985). Studies of CBT for people with early RA have found improvements in health outcomes both in the short-term (Parker *et al.*, 1995) and long-term (Sharpe *et al.*, 2001b). As none of these studies included a baseline phase it is also not possible to be certain that improvements resulted from the intervention rather than pre-group sensitization.

With respect to Hypothesis 1.2, there was more evidence of change in adaptive self-regulatory processes than health outcomes following self-management intervention. There was a significant increase in self-efficacy for pain and self-efficacy for other arthritis symptoms in the treatment group but not the control group. The baseline phase was stable and the improvement occurred following the intervention. The improvements were maintained at 3-month and 9-month follow-up, although there was a small reduction in the treatment group's mean scores. While it could be argued that changes in self-efficacy may be attributed to attention and support, it seems highly unlikely that these improvements could be associated with non-specific effects. This is particularly so given the failure to find similar effects in trials with an attention placebo group (Parker *et al.*, 1995).

There was a significant increase in the use of planning as a coping strategy in the treatment group but not the control group, shown by a significant interaction effect on the ANOVA and ANCOVA. The baseline phase was stable and there was a significant increase in the use of planning in both groups following the intervention. However, the treatment group showed a much larger increase. This is maintained at 3-month follow-up but not the 9-month follow-up. This indicates that following a self-management intervention people make greater use of planning as a coping strategy and continue to do this for at least three months.

The planned contrasts also showed significantly greater short-term adaptive changes in illness identity, perceived consequences and the use of strengthening exercises, in the

treatment group than in the control group, following the intervention. There was also a significant increase in the practice of stretching exercises and relaxation in the whole sample. Most of the improvement, in these self-management behaviours, occurred in the treatment group immediately following the intervention and was maintained at 3 and 9-month follow-up. The small positive changes in practice of self-management behaviours in the control group may have arisen from a Hawthorne effect (i.e. knowing that one is being monitored).

In summary, the treatment group showed significantly greater adaptive changes in self-regulatory variables than the control group. Immediately following the intervention the treatment group had significantly greater decreases in illness identity and perceived consequences, and increases in self-efficacy for pain, self-efficacy for other arthritis symptoms, use of planning as a coping strategy and practice of strengthening exercises, than the control group. The changes in illness identity, self-efficacy for pain, self-efficacy for other arthritis symptoms, use of planning as a coping strategy and practice of strengthening exercises were maintained at 3 month follow up. The improvements in self-efficacy for pain and other arthritis symptoms and practice of strengthening exercises were also maintained at 9-month follow up. For illness identity some of the improvement occurred during the baseline phase but for the other variables the improvements occurred during the intervention phase.

As discussed in Chapter 3, improvements in self-efficacy, coping and self-management behaviours have also been reported in several studies evaluating the ASMP (Boutaugh & Lorig, 1996) and psychological interventions for people with RA (Astin *et al.*, 2002). However, this is the first time that adaptive changes in illness representations (illness identity and consequences) following psycho-educational intervention for chronic RA have been reported.

There is evidence that changing self-efficacy or confidence in one's ability to manage the illness, seems to be an important mechanism by which psycho-educational interventions assist people to improve self-regulation of rheumatic disease (Lorig & Gonzalez, 1992;

Smarr *et al.*, 1997). The present study, and other studies (Germond *et al.*, 1993; Kraimaat *et al.*, 1995c; Parker *et al.*, 1988b; Smith *et al.*, 1988), have found significant changes in self-efficacy, coping and self-management behaviours, without sustained changes in pain, disability or depression. This suggests that while these variables may be important, change in these variables is not always sufficient to produce sustained change in health outcomes and that other variables are also likely to be important mediators of change.

The findings of the present study, and review of the literature, suggest that both CBT and self-management interventions are less effective for hospital out-patients with chronic and severe RA than for people with early RA or people with RA recruited from community populations. It is important to understand why this might be in order to improve interventions and health outcomes for people with chronic RA. There are a number of possible explanations for these findings.

First, it is possible that differences in the type of intervention may explain the results. Studies with mixed populations predominantly used comprehensive self-management interventions and some studies with RA samples used more focused CBT interventions that may have omitted some key treatment components. However, the present study used a comprehensive self-management intervention, and similar to Taal *et al.*, (1993b) and O'Leary *et al.*, (1988) did not demonstrate sustained changes in pain, disability or depression. The most important difference between the intervention used in the present study and those used in other studies of the ASMP was that it was delivered primarily by health professionals supported by co-leaders with RA. However, studies have shown that the ASMP is equally effective when delivered by health professionals or lay leaders (Cohen *et al.*, 1986; Lorig *et al.*, 1986). Therefore, it seems unlikely that intervention methods alone could account for differences in outcome between studies of the ASMP and the present study, or studies of CBT for people with RA.

Second the sample size in the present study, and studies of CBT for RA populations, was smaller than that used in studies of the ASMP. This increases the likelihood that clinically

meaningful effects may not have been statistically evident, a Type II error, this will be discussed further in section 10.5.

Third, the findings may reflect differences in the populations investigated, with respect to diagnosis, severity or chronicity of illness. Studies of the ASMP have generally investigated community samples of people with arthritis, mainly OA, but with a significant number of people who report they have RA. As RA is a more serious condition, with higher levels of pain and disability, it is possible that it is more difficult to influence behaviour and health status in RA than in OA. Barlow *et al.*, (2000) investigated this in a RCT of the ASMP in a UK community population of people with arthritis (mainly OA and RA). At 4-month follow-up improvements in emotional state, self-efficacy and self-management behaviours were independent of the type of arthritis. Therefore, diagnosis alone may not explain the differences between results.

Severity of RA may be a factor in explaining the findings. The ASMP studies primarily used community samples and the CBT interventions used samples drawn from hospital clinics who are known to have more serious disease, poorer function and prognosis (Buckley, 1997). Participants in the present study had relatively high levels of pain and physical disability compared to other studies of chronic RA. More severe RA (greater pain, fatigue, and physical disability, frequent flares and progression of the disease) may limit people's ability to successfully manage RA and benefit from psycho-educational intervention (Kraaimaat *et al.*, 1995c; Parker *et al.*, 1988b).

There is also evidence that psycho-educational interventions may be more effective for people with a shorter duration of RA (Astin *et al.*, 2002). People in the present study had long-standing RA (mean duration=13.4 years). It might be argued that, for people with chronic RA, maladaptive self-regulatory processes will have become so firmly established that brief interventions are not sufficient to develop and maintain new adaptive patterns. However, contrary to this, both the present study and other studies have shown adaptive changes in illness representations, self-efficacy, coping procedures and self-management behaviours in people with chronic RA following psycho-educational

intervention (Astin *et al.*, 2002). As discussed in Chapter 2, these factors have also been associated with better health outcomes (Huyser & Parker, 2002; Keefe *et al.*, 2002).

The evidence seems to suggest that psycho-educational interventions can change self-regulatory processes and health outcomes in chronic RA but that maintaining these improvements is more problematic. There is evidence that when adaptive changes in self-regulatory processes are sustained this can lead to better long-term health outcomes. For example, continued daily use of cognitive-behavioural coping strategies was associated with better long-term outcomes following a CBT intervention for chronic RA (Parker *et al.*, 1988b). It is possible that the fluctuating pattern of disease activity in RA may make maintaining adaptive changes particularly difficult. When people with chronic RA are faced with disease flares, or other life stressors, they may perceive their illness to be overwhelming. This may lead them to curtail effective coping efforts and adopt counterproductive strategies, thereby precipitating greater increases in pain, distress, and disability. Times when RA is less active, and symptoms diminish, may also trigger unhelpful changes in people's illness and treatment representations. At such times, people may reduce their practice of cognitive-behavioural coping skills, thereby setting the stage for maladaptive coping if symptoms intensify. People may also discontinue active attempts to self-regulate their illness (e.g. practice of physical exercises) if they produce short-term increases in their symptoms. Such increases may be appraised, as evidence of increased disease activity, that attempts at self-management are ineffective or even that the illness is uncontrollable. Relapse prevention is intended to counteract these processes (Huyser & Parker, 2002). It seems likely that barriers to initiating and maintaining change in people with severe and chronic RA are greater but further work is needed to explore these. In the present study, although relapse prevention was briefly addressed, other studies have used much more extensive relapse prevention methods (Parker *et al.*, 1995). Overall, it seems likely that for people with severe and chronic RA more intensive interventions, with regular booster sessions to maintain change may be needed (Parker *et al.*, 1995). This parallels conclusions that have been drawn about the effectiveness of psychological treatments for people with chronic pain (Morley, Eccleston & Williams, 1999).

Fourth, it is possible that differences in study design and methodology might explain the differences in findings. As discussed in Chapter 3 the method of recruitment employed in most ASMP studies may produce a selection bias. Participants are likely to be a subset of the total population of people with arthritis, who are more motivated to participate actively in the management of their arthritis. Motivational factors are critical to the success of psycho-educational interventions. It seems likely that the recruitment methods used in the present study selected a more representative group of people with chronic RA, who may have been less motivated to engage in self-management, and who therefore benefited less from the intervention. This factor would have been exacerbated by the intention-to-treat analysis, which included all participants from the treatment arm of the study in the analyses even if they did not start or complete the intervention. Many of the studies of the ASMP have used pragmatic analyses, which are likely to produce apparently larger treatment effects (e.g. Barlow *et al.*, 2000).

It remains unclear how effective the ASMP would be for people with arthritis who are less motivated. Different recruitment strategies and research designs are needed in order to explore the influence of motivational factors in known populations with arthritis (Barlow *et al.*, 2002b).

Unlike the present study and other studies of CBT for RA, most studies of the ASMP, used a waiting list control group design. Long-term results are based on within group analyses which makes interpretation of the longitudinal phase of ASMP studies difficult, especially given the recruitment methods usually employed in these studies. People may be more likely to volunteer to attend a self-management programme when they are experiencing more difficulty managing their arthritis. Enrolment during periods of heightened disease activity or severity might influence the apparent effect size of the intervention. In non-controlled studies regression to the mean may produce apparent treatment effects when, in reality, the effect of treatment is small or non-existent (Hawley, 1995). This is particularly important as the most notable difference between the results of studies of the ASMP and the present study, and other studies of CBT for RA, is in the long-term follow-up.

Overall, the results of this study indicate that the most likely explanation for the differences between the findings of self-management and CBT studies is not the type of intervention but rather differences in the populations studied and the designs of the studies. The present study provides some evidence that a self-management intervention for hospital out-patients with chronic RA may produce some short-term improvements in pain and depression and long-term improvements in individualized quality of life. There is stronger evidence that the intervention produced short-term improvements in illness identity, perceived consequences and use of planning as a coping strategy, and long-term improvements in self-efficacy for pain and other arthritis symptoms and practice of strengthening exercises.

10.3 The relationship between illness representations, coping and health outcomes in chronic RA

The analyses, in the pooled sample, explore the relationships between self-regulatory processes and health outcomes in chronic RA. Four issues were examined: 1. the relationships between illness representations and coping procedures at baseline (hypothesis 2.1); 2. whether illness representations and coping procedures account for significant variance in health outcomes at baseline (Hypothesis 2.2); 3. whether initial illness representations and coping procedures predict future health outcomes at 3, 6 and 12 months (Hypothesis 2.3); 4. whether changes in illness representations and coping procedures explain health outcomes at 3, 6 and 12 months (Hypothesis 2.4). Given the number of analyses only the key findings will be discussed.

10.3.1 Relationships between illness representations and coping

With respect to Hypothesis 2.1 all the significant associations between illness representations and coping procedures were in the predicted directions. This is in line with the SRM which proposes that illness representations shape coping efforts. A consistent pattern emerged. Identifying fewer symptoms as part of RA, perceiving RA symptoms to be more coherent and the consequences of RA to be less serious were all associated with: 1.

greater use of the adaptive coping strategy, Positive Reinterpretation/Growth; 2. less use of the maladaptive strategy, Behavioural Disengagement. The strongest relationships were found with Behavioural Disengagement. The first finding suggests that when people perceive their illness is less severe (fewer and more coherent symptoms and less serious consequences) they tend to make greater use of cognitive strategies which may help them re-appraise the significance of their condition, although the direction of causality cannot be determined from these correlational analyses. The second finding suggests that perceiving RA as having multiple incoherent symptoms and serious consequences is associated with passive coping, giving up attempts to achieve life goals and engage in valued activities. This finding replicates the study by Scharloo *et al.* (1999) who found in chronic RA that greater perceived consequences were associated with more passive coping. This strategy may assist people in tolerating episodes of uncontrollable disease activity. However, when the disease is less active, it may be associated with the perception that active coping is not effective and greater emotional distress. Such a state of learned helplessness could be created when an illness such as RA is repeatedly experienced as uncontrollable (Affleck *et al.*, 1987b).

The present study also found that perceiving RA as more controllable was associated with greater use of adaptive coping strategies, Active Coping and Planning. This is logical as people are likely to make active attempts to manage the illness if it is perceived to be controllable and coping efforts are perceived to be efficacious and easy to perform. The same finding has been reported in other studies of arthritis (Orbell *et al.*, 1998; Schiaffino, Shawaryn & Blum 1998). These results are also consistent with work in other illnesses. For example, Moss-Morris *et al.*, (1996) studied the role of illness representations in people coping with chronic fatigue syndrome (CFS). The study revealed that the identity and cure/ control dimensions were significantly correlated with active coping, seeking social support and behavioural disengagement. People who perceived that their illness had serious consequences had higher scores on denial and behavioural disengagement coping subscales.

A recent meta-analysis of 45 studies of chronic illness guided by Leventhal's SRM (Hagger & Orbell, 2003), found theoretically consistent patterns of relationships between illness representations and coping across studies. In line with the results of the present study, perceptions of serious consequences and a strong illness identity were positively associated with the use of coping strategies of expressing emotions and avoidance/ denial. Control/ cure perceptions were positively associated with cognitive reappraisal, problem-focused coping- generic and seeking social support.

10.3.2 Do illness representations and coping explain health outcomes at baseline?

In the cross-sectional analyses the entire regressions were able to account for a large amount of the variance in pain, physical disability, emotional distress and individualized quality of life. They explained a significant but smaller amount of variance in social and occupational function, disease activity and health-care utilization.

These results compare favourably with other studies. For example, in a large cross-sectional study of people with RA, Strahl, Kleinknecht and Dinnel (2000) found that a combination of socio-demographic, physical and psychological variables accounted for between 9-38% of the variance in the five AIMS2 scales. In the present study, the analyses explained between 17-71.4% of the variance in the same measures.

With respect to Hypothesis 2.2, illness representations and coping procedures accounted for a significant amount of variance in health outcomes (pain, disability, emotional distress, social function and disease activity) at baseline. These results were obtained even after controlling for demographic factors, disease activity and severity, physical, emotional and social function. In 10 of 20 analyses illness representation, coping and emotional variables explained more variance in health outcomes than socio-demographic, disease activity, pain, and physical and social function variables combined. This emphasizes the importance of self-regulatory processes in RA. It confirms previous work showing that psychosocial factors explain more variance in health outcomes than objective measures of disease activity

(Young, 1992). It is particularly impressive because socio-demographic factors, subjective pain, physical and social function were also controlled for in the analyses.

There were three primary outcomes in the present study, pain, disability and emotional distress, which will be considered in more detail. Illness representations and coping procedures only accounted for a relatively modest amount of additional variance in pain (6.2-13.3%) in 3 of 5 analyses. However, In 4 of 5 analyses illness representations, coping procedures and emotional state accounted for more variance in pain than socio-demographic factors, disease activity, physical and social function. This is consistent with findings from other studies showing that in RA psychosocial variables account for more variance in pain than measures of disease activity (Parker *et al.*, 1988a). Lower pain was most consistently predicted by identifying fewer symptoms as part of RA (lower IPQ Identity) and greater self-efficacy for functional activities. Much of the variance in pain remains unexplained. Further research might investigate other factors that could explain pain perception in RA (e.g. personality, stress, attention, pain anxiety; Kludt, 2000; Strahl *et al.*, 2000).

Self-regulatory variables accounted for a large amount of the variance in physical disability (26.3-51.8%). The strongest and most consistent predictor of lower disability was greater self-efficacy for functional activities. However, there may be a degree of overlap between confidence in performance of functional activities and self-reported ability to perform functional activities. Greater self-efficacy might lead to endorsement of better functional status even when function does not change (Hawley, 1995).

With respect to measures of emotional distress, illness representations and coping procedures explained a large amount of the variance (26.3%) in depression (HADS). People who perceive the consequences of their RA to be more serious, Cope by focusing more on dealing with their RA and giving up attempts to achieve other goals and valued activities, and report lower social activity and support feel more depressed. These findings are consistent with other studies that have found that perceiving the consequences of RA as more serious (Murphy *et al.*, 1999), the loss of valued activities

(Katz & Yelin, 1995) and lower social support (Kraaimaat *et al.*, 1995a) are strongly associated with depression in RA.

The association between higher levels of depression and perceiving the consequences of RA to be more serious was found even though disease activity, pain and disability were controlled for. This provides evidence that greater perceived consequences do not simply reflect the fact that RA is actually more serious.

The regression explained a large amount, 60.3%, of the variance in anxiety (HADS).

One interesting relationship that emerged was that lower anxiety was explained by higher disease activity (ESR). This was the only immunological measure explaining unique variance in health outcomes found in all the cross-sectional analyses. The finding that lower disease activity (ESR) was associated with high anxiety in both the preliminary correlational and multiple regression analysis emphasizes the dissociation between physical and psychological outcomes in RA. However, it was unexpected as previous studies have either found weak positive associations between other measures of disease activity (e.g. joint counts) and emotional distress (Parker *et al.*, 1991; Parker *et al.*, 1992) or no association between measures of disease activity and anxiety (Eberhardt, Larsson, Nived 1993). It is possible that a third variable, such as age, might account for the relationship between ESR and anxiety, as ESR increases with age and higher levels of anxiety have been found in younger people with RA (Pimm, 1989). However, age was entered in this regression and so this could not have accounted for the association between ESR and anxiety. There is some evidence that levels of anxiety are higher and disease activity lower in people with sero-negative RA (Kludt, 2000) and this might have accounted for the finding. However, the partial correlation between ESR and anxiety, controlling for rheumatoid factor, was still significant ($r = -.24^*$). Another possible explanation might be that in a chronic fluctuating illness like RA a period of time when the disease is less active may increase anxiety because it heightens awareness of the impact of the condition on the person's life during periods of high disease activity and anticipation of future worsening in health. An alternative explanation might be that people with RA who have higher levels of anxiety may be more likely to be attending

hospital out-patient clinics even when their disease is relatively less active. Levels of emotional distress in people with RA are higher in studies of hospital clinic than community populations (DeVellis, 1993). In addition, anxiety has been found to be an important predictor of help-seeking behaviour (Cameron, Leventhal & Love, 1998). In a longitudinal study of women with breast cancer in remission, Cameron *et al.* (1998) found that anxiety was associated with the activation of illness representations that triggered attention to sensations worry and protective coping (seeking medical help) in response to a somatic cue. A similar process could lead to people with relatively less active RA, but higher levels of anxiety, being over-represented in hospital out-patient clinic populations.

Illness representations and coping procedures explained a large amount of the variance in anxiety (29.1%). People who perceive their RA as having more frequent and incoherent symptoms and to be less controllable were more anxious. Those who cope more by giving up attempts to achieve other goals and valued activities and focussing on expressing their emotions and less by accepting RA were also more anxious. Psychosocial factors have been found to be associated with emotional distress in RA in previous studies (Bradley & Alberts, 1999). However, these results are interesting because relatively few studies have investigated correlates of anxiety in RA (Newman & Mulligan, 2000) and even fewer the relationships between illness representations and anxiety.

These analyses indicate the strong association between self-regulatory processes and health outcomes in RA. However, as these findings are based on cross-sectional analyses, it is not possible to make causal statements about these relationships. For example, people may view their illness more negatively because they are depressed, or people may become depressed because they view their illness so seriously and feel they have no control over it. The longitudinal analyses allow the direction of causal relationships to be explored further.

10.3.3 Do illness representations and coping predict future health outcomes?

In the longitudinal analyses the entire regressions were able to account for a large amount of the variance in all health outcomes, except health-care utilization where they predicted a significant, but smaller, amount (17.6-31.5%). These results compare favourably with other longitudinal studies of RA (Scharloo *et al.*, 1999; Uhlig *et al.*, 2000).

With respect to Hypothesis 2.3 Baseline illness representations and coping procedures predicted a significant amount of variance in subsequent health outcomes (pain, disability, emotional distress, disease activity and health care utilization) at 3, 6 and/or 12 months.

Overall, the amount of variance explained was small, usually less than 10% (range=2.1-17.8%) and was only found in 16 of 74 analyses. However, these results were found after controlling for the baseline of the dependent variable, socio-demographic factors, disease activity, physical, social and emotional function, and up to a year later. The amount of variance explained by illness representations and coping procedures was similar to that reported by Scharloo *et al.*, (1999) in their longitudinal study of chronic RA.

When baseline illness representations and coping procedures did explain significant additional variance in health outcomes the relationships tended to be reasonably consistent over time. For six outcome measures (pain distress, HAQ, AIMS2 Physical, CRP, EMS and doctor visits) they predicted significant additional variance in two or more of the three analyses.

In addition, some consistent relationships were found between particular self-regulatory variables and health outcomes. Greater practice of stretching exercises at baseline predicted lower pain distress at 6 and 12 months. Previous studies have shown that psychosocial factors are strongly associated with pain (Bradley & Alberts, 1999; Buescher *et al.*, 1991; Newman *et al.*, 1990). This finding suggests that engaging in active self-management strategies is important for regulation of subsequent emotional responses to symptoms.

Although the strong association between initial and subsequent measurements of the HAQ and AIMS2 Physical leave little room for explaining variance, baseline self-regulatory variables consistently added small but significant amounts of variance. Greater generalized self-efficacy and self-efficacy for functional activities were the most consistent baseline self-regulatory variables predicting subsequent physical disability. This is in agreement with other studies showing that psychosocial factors predict physical disability (Scott *et al.*, 2000; Uhlig *et al.*, 2000; Wolfe, 2000). More specifically, associations between self-efficacy and physical functioning have also been reported previously (Lorig *et al.*, 1989a; O'Leary *et al.*, 1988). However, as mentioned earlier, there is probably some overlap between the self-report measures of physical disability and the function sub-scale of the Arthritis Self-Efficacy Scale.

Greater self-efficacy for functional activities and less practice of relaxation exercises at baseline predicted lower disease activity (CRP) at 3 and 6 months. O'Leary *et al.* (1988) also found associations between self-efficacy and measures of disease activity in people with RA. The potential for psychological factors to influence immunological function and subsequently disease activity in RA has been an area of increasing research interest in the field of psychoimmunology (Huyser & Parker, 1998; McFarlane & Brooks, 1990). For example, Horton-Ausknecht, Mitzdorf, and Melchart, (2000) found, in people with RA, that hypnosis, using specific autoimmune related imagery, produced clinically significant changes in disease activity (ESR) when compared to relaxation and waiting-list control groups. Within the context of the SRM these findings also emphasize the point that self-regulatory processes are not only influenced by disease activity, but that the relationship may be bi-directional.

Interpretation of the findings for relaxation are complex. It is difficult to postulate a mechanism by which greater practice of relaxation might increase future disease activity. Although the analyses are longitudinal causal relationships cannot be assumed, as other explanations for the findings are possible. For example, a third factor, such as disease severity, might influence both practice of relaxation at baseline and subsequent CRP. At baseline, before participants are taught formal relaxation techniques, they may interpret

the question as meaning "how often do you try to relax or rest?". People with more severe disease at baseline are more likely to have to rest and so will report greater use of relaxation. Although baseline levels of CRP are controlled for in the analysis these are likely to explain only part of the variance in subsequent disease activity. Other measures of disease activity and severity were excluded from the analysis on theoretical grounds, as they are measuring the same construct as CRP. Therefore, more severe disease at baseline could predict higher CRP at subsequent assessments. An alternative, more psychological explanation, might be that people who perceive their RA as more severe may make greater use of passive avoidant coping strategies, such as rest. Passive coping may lead to less effective management of RA or stress, producing greater subsequent disease activity. Given the association between lower disease activity (ESR) and higher anxiety, identified in the cross-sectional analysis, it was also interesting that higher baseline anxiety predicted lower disease activity (CRP) at time 4. This tends to support the suggestion made earlier that anxiety may trigger attempts to actively manage RA leading to subsequent improvements in disease activity.

The use of coping strategies at baseline predicted subsequent health-care utilization. Greater use of restraint coping predicted lower frequency of doctor visits at 6 months and less use of positive interpretation/growth predicted lower frequency of doctor visits at 12 months. People who cope with RA by restraining themselves from taking any action until the situation improves not surprisingly make fewer visits to their doctor. People who cope with their RA by viewing their situation from a different perspective and identifying positive features of their situation make more visits to the doctor a year later. Within the dynamic processes proposed by the SRM one possible interpretation of this finding is that those who try to cope in this way, and who are then faced with the realities of a serious chronic illness, begin to recognise that they need to tackle their problems more actively and so seek further help. Consistent with this explanation is the finding, by Scharloo *et al.* (1999), that perceiving the consequences of RA as more serious is associated with greater use of hospital out-patient services a year later.

10.3.4 Do changes in illness representations and coping predict health outcomes?

With respect to Hypothesis 2.4 change in illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social and occupational function, quality of life and disease activity) at 3, 6 and/or 12 months, illness representations and coping procedures most consistently predicted pain, disability, emotional distress, social function and quality of life. Overall a small to moderate amount of variance was explained (range=1.9-24.5%) but the results were consistent being found in 51 of 74 analyses. Illness representations and coping procedures explained the greatest average amount of variance in pain (10-17.4%), emotional distress (11.3%) and individualized quality of life (11.1%). Illness representations and coping procedures were stronger and more consistent predictors of health outcomes when measured concurrently rather than at baseline. The results are impressive as they were found after controlling for the baseline of the dependent variable, socio-demographic factors, disease activity, physical, social and emotional function, and baseline illness representations and coping procedures. Apart from the baseline of the dependent variable, concurrent illness representations and coping procedures were the most important predictors of health outcomes.

These results are of particular interest as few other studies have examined self-regulatory predictors of health outcomes at multiple time points or investigated how change in self-regulatory variables affect health outcomes. Sharpe *et al.*, (2001a) note that prospective studies assessing relationships between factors over long time periods are likely to miss the subtle interaction between self-regulatory processes that occur over relatively short periods of time. The findings that concurrent self-regulatory variables are stronger and more consistent predictors of health outcomes is consistent with the dynamic nature of the relationships between self-regulatory processes and health outcomes in RA.

Controlling for baseline scores, a number of specific concurrent self-regulatory variables were found to consistently predict particular health outcomes. Greater pain was

consistently predicted by increases in the perceived range and frequency of RA symptoms, increases in the perceived seriousness of the consequences of RA, decreased confidence to manage pain and increased use of passive coping over time. These findings are consistent with other studies showing that psychosocial factors are strongly associated with pain (Bradley & Alberts, 1999; Buescher *et al.*, 1991; Newman *et al.*, 1990).

Greater disability was predicted by increases in the perceived range and frequency of RA symptoms and decreased confidence to manage pain and perform functional activities over time. These findings are consistent with the analyses of cross-sectional relationships and baseline predictors, but provide stronger evidence that changes in symptom perception and confidence to manage pain and perform functional activities influence people's self-reported physical disability. As discussed earlier, in relation to the baseline predictors of physical disability, these findings are in agreement with other studies showing that psychosocial factors predict physical disability (Scott *et al.*, 2000; Uhlig *et al.*, 2000; Wolfe, 2000). More specifically, associations between self-efficacy and physical functioning have also been reported previously (Lorig *et al.*, 1989a; O'Leary *et al.*, 1988). However, as mentioned earlier, there is probably some overlap between the self-report measures of physical disability and the function sub-scale of the Arthritis Self-Efficacy Scale.

Greater emotional distress was predicted by increases in the perceived range and frequency of RA symptoms, decreased confidence to manage other arthritis symptoms (e.g. fatigue, frustration and low mood), and increased use of emotion focused coping over time. This is consistent with other studies that have shown that illness related cognitions and coping strategies predict emotional distress in RA (Bradley & Alberts, 1999; Wright *et al.*, 1996).

Several self-regulatory variables were found to predict more than one health outcome. Increases in the perceived range and frequency of RA symptoms (illness identity) were found to consistently predict greater pain, physical disability, emotional distress and poorer individualized quality of life. This is consistent with Scharloo *et al.* (1999) who

found, in RA, that a stronger illness identity, at baseline, was associated with more pain, tiredness, and depression one year later. Kaptein *et al.*, (2003) has recently reviewed studies of illness representations in chronic illness. They found that a stronger illness identity was associated with negative health outcomes in studies of all five chronic illnesses they reviewed (asthma, chronic obstructive pulmonary disease, COPD, cardiovascular disorders, cancer and neurological conditions). The relationship between illness identity and individualized quality of life is of particular note as it has not been reported previously and individualized quality of life was found to change significantly following the self-management intervention. This suggests that symptom perception may be an important target for psycho-educational intervention in RA.

It is important to note that these relationships could be confounded by measurement artefacts. For example, illness identity may be indicative of symptom experience rather than beliefs about the symptoms associated with the illness in question because identity was measured using symptom reports. It could be argued that these associations between illness identity and illness outcomes may only reflect relationships relevant to the person's current experiences with the illness which could be confounded by illness state (Hagger & Orbell, 2003). Alternatively, it has been argued that the IPQ Identity scale is more than just a proxy measure of disease activity or severity (Moss-Morris *et al.*, 2002). The results of the cross-sectional analyses, in which illness identity explained health outcomes, even when disease activity, pain, physical function and emotional state were controlled for, support this contention.

Increases in the perceived consequences of RA predicted greater pain (at 3 months and especially at 12 months) and longer duration of early morning stiffness (at 6 and 12 months). This suggests that changes in people's representations of their illness influence perception of symptoms, in terms of range, frequency and duration. Increases in perceived consequences were also found, less consistently, to predict worse physical disability, occupational function and individualized quality of life. It was interesting that perceived consequences did not predict depression, as it did in the cross-sectional analysis and in other cross-sectional studies (Murphey *et al.*, 1999). These findings are

complementary to those reported in other studies. Perceiving RA as having more serious consequences at baseline has been shown to predict more visits to the outpatient clinic, more tiredness, and higher anxiety scores a year later Scharloo *et al.*, 1999; and greater depression 21 months later (Sharpe *et al.* 2001a). Kaptein *et al.* (2003) found in their review that greater perceived consequences had been associated with negative health outcomes in studies of asthma, COPD and cardiovascular disease.

These results are consistent with Hagger and Orbell (2003) meta-analytic review. They found that a strong illness identity and belief that the illness has serious consequences were negatively related to adaptive illness outcomes (physical, social and role functioning, vitality, and psychological well-being) but positively related to the maladaptive outcome of psychological distress.

Hagger and Orbell (2003) also found that perceiving the illness as curable/ controllable was significantly and positively related to the adaptive outcomes of psychological well-being, social functioning and vitality and negatively related to psychological distress and disease state. In the present study, the IPQ Control/Cure scale did not emerge as a strong predictor of health outcomes. One possible explanation for this was the inclusion of the Arthritis Self-efficacy Scale which may assess similar constructs in the domain of perceived controllability. Weinman *et al.*, (1996) found that the IPQ Control/Cure scale was measuring two independent factors 1. Personal control and self-efficacy, and 2. Beliefs about the efficacy of treatment. (see section 10.5.7). Similarly, the Arthritis Self-efficacy Scale may not only measure self-efficacy. Many of the items seem to be tapping both self-efficacy beliefs and outcome expectancies e.g. "How certain are you that you can decrease your pain quite a bit? and "; "How certain are you that you can control your fatigue?" (see section 10.6.1 for further discussion of this issue).

Increased self-efficacy for pain predicted lower pain and physical disability. Increased Self-efficacy for functional activities predicted lower physical disability and increased self-efficacy for other arthritis symptoms predicted lower emotional distress. Therefore confidence in ones ability to perform behaviours to manage a specific health outcome

tended to predict better subsequent scores for that specific health outcome. For example, the majority of items on the self-efficacy for other arthritis symptoms sub-scale address confidence in one's ability to manage symptoms characteristic of emotional distress (fatigue, frustration, low mood and engagement in enjoyable activities). These results confirm the findings of other studies indicating that self-efficacy is an important factor in adaptation to rheumatic disease (Barlow *et al.*, 1996; Daltroy & Liang, 1993; Lorig *et al.*, 1989b; Manne & Zautra, 1992; Parker & Wright, 1995).

Coping procedures were also found to predict health outcomes in a few analyses. Increased use of passive (restraint coping) and emotion focused (focusing on and venting emotions) coping and decreased use of active coping (increased behaviour) predicted worse pain, emotional distress and social function respectively. This is consistent with the literature on coping with chronic painful conditions in general and RA specifically. Studies have found that coping is associated with pain, psychological distress, and physical disability (Bradley & Alberts, 1999; Brown & Nicassio, 1987; Jensen *et al.*, 1991; Keefe *et al.*, 1992; Newman *et al.*, 1990; Young, 1992).

Comparing the baseline and concurrent predictors an interesting pattern emerges. It appears that at baseline self-efficacy, coping strategies and self-management behaviours were the strongest and most consistent self-regulatory variables predicting future health outcomes. They predicted far fewer outcomes but these tended to be the more objective ones of disease activity and doctor visits. Changes in illness representations were stronger and more consistent predictors of health outcomes, especially the more subjective outcomes (pain, emotional distress, social function and individualized quality of life). Tentatively, these findings could be interpreted as consistent with the self-regulatory processes proposed by the SRM. Changes in people's illness representations would be expected to have a more direct or immediate effect on variables like symptom experience and emotional state. The influence of illness representations on more objective or behavioural outcomes is likely to be less immediate as it is assumed to be mediated by coping efforts.

Although interesting relationships emerged in the prospective analyses, it is notable that in most analyses the amount of variance which was accounted for by the baseline measurement of the dependent variable was very high. It may be that these stable patterns are established very early in the development of the illness. For example, Sharpe (1999) found similar relationships in a longitudinal study of people with early RA, with an average duration of one year. Given this, these relationships will remain difficult to elucidate in longitudinal studies. Intervention studies may be more useful for exploring these relationships.

10.4 Do illness representations or coping procedures moderate or mediate the effects of a self-management intervention

Analyses were conducted to determine whether self-regulatory variables moderated or mediated the improvements in individualized quality of life and self-efficacy found following self-management intervention. As discussed above this should: 1. aid identification of those people who are most/least likely to benefit from intervention. 2. increase understanding of the mechanisms of change through which treatment is effective. 3. Lead to suggestions for developing treatments to maximise effectiveness.

There were two interesting findings from the analyses examining moderators of treatment outcome. First, people with greater confidence in their ability to manage pain, at the beginning of the study, who then received a self-management intervention, had greater improvement in individualized quality of life at the 9-month follow-up. Those who are most confident in their ability to manage pain before attending the self-management programme seem to be most likely to maintain improvements in individualized quality of life achieved during the intervention. One possible interpretation of this is that people who are more confident about managing pain may persist with new approaches to self-management despite setbacks (e.g. flares of disease activity) and so in the long-term reduce the impact of RA on valued areas of their life. These results are consistent with studies that have shown that self-efficacy is an important predictor of future health outcomes in rheumatic disease (Boutaugh & Lorig, 1996).

There is also evidence that changes in self-efficacy are an important part of the mechanism in the process of change in health outcomes occurring during self-management intervention (Boutaugh & Lorig, 1996). However, little is known about the processes underlying changes in self-efficacy and whether other factors influence this process (Lorig & Gonzalez, 1992). Recent descriptions of both Leventhal's SRM and other self-regulatory models, such as the Carver and Scheier model, have identified that confidence can be an important factor in the process of self regulation of health and illness, especially when predicting complex behaviours, such as exercise (Cameron & Leventhal, 2003; Scheier & Carver, 2003). The SRM suggests that perceptions of ones ability to perform coping behaviours should be influenced by cognitive representations of the illness and appraisals of coping efforts. The moderator and mediator analyses enable examination of whether illness representations or coping procedures influence changes in self-efficacy occurring during the self-management intervention.

With respect to these issues, the second significant finding from the moderator analyses, was that people who made greater use of ignoring sensation as a pain coping strategy, at the beginning of the study, and then received a self-management intervention, had greater improvements in self-efficacy for other arthritis symptoms at the 3 and 9-month follow-up.

One possible interpretation of these results is that attempting to ignore sensations while putting new approaches to self-management (e.g. increased physical exercise) into effect may enable people to tolerate the likely short-term increases in symptoms associated with the new behaviours, or other setbacks, and persist with new behaviours which in turn lead to increased confidence to manage symptoms and continue valued activities.

It is interesting that ignoring sensations was the most frequently used pain coping strategy in the whole sample at baseline. This finding is consistent with the dynamic nature of self-regulatory processes. The moderator analyses indicate that greater use of ignoring sensation while learning how to manage RA subsequently results in greater confidence to manage RA symptoms. Within the SRM, appraisal processes could provide a mechanism

explaining this finding. If the person evaluates ignoring sensations as a coping strategy that enables them to achieve their goals (e.g. reduce symptoms and continue valued activities) then this could lead to changes in their illness representation through feedback processes. Such a mechanism operating during the process of adaptation to RA could have led to people making greater use of this coping strategy.

This study also confirms in a causal analysis previous work that suggested that ignoring pain sensation was associated with self-efficacy. In a cross-sectional study of people with OA of the knee (Keefe *et al.*, 1997b) found that ignoring sensations (assessed on the CSQ) explained a significant amount of variance in self-efficacy for pain (assessed on the ASE scale). As in the present study greater use of ignoring sensations was associated with higher self-efficacy.

Self-regulatory variables only moderated treatment outcome at follow-up. Therefore it is possible that initially high levels of self-efficacy for pain and use of ignoring sensations as a pain coping strategy are important for maintaining treatment gains rather than producing changes during intervention. Maintaining the treatment gains from psycho-educational intervention for people with chronic RA is problematic. The findings that self-regulatory variables may influence people's ability to maintain treatment gains is therefore important. Modification of interventions to enhance self-regulatory processes that may assist in maintaining gains should be explored.

The mediator analyses also produced some very interesting results. Reductions in the perceived range and frequency of symptoms attributed to RA (illness identity) during the self-management intervention mediated all three of the main benefits of treatment (individualized quality of life, self-efficacy for pain and other arthritis symptoms). In addition, increases in the perceived controllability of RA during treatment mediated the improvement in self-efficacy for other arthritis symptoms found immediately after treatment.

Rhee *et al.*, (2000) also investigated mediators of treatment outcome. They re-examined the Parker *et al.*, (1995) study, using path analysis to test whether changes in cognitions and coping strategies, mediate between cognitive behaviour therapy and reduced pain and depression in early RA patients. They found that together helplessness (AHI), self-efficacy (ASE scale) and confidence to manage pain (PCRT factor from the CSQ) mediated the immediate effects of the stress management intervention on pain and depression. They recommended that psycho-educational interventions should be refined to facilitate greater change in these variables. However, the results of the present study suggest that improvements in quality of life were mediated by changes in illness identity rather than self-efficacy. This suggests that changes in self-efficacy may not be a necessary condition for improvement in all health outcomes. Although, it is important to note that the moderator analyses did demonstrate the importance of initial levels of self-efficacy in maintaining the improvements in quality of life resulting from treatment.

The results of the mediator analyses suggest that illness representations play a role in the process of change during self-management intervention. Reducing the perceived range and frequency of RA symptoms seems to be an important mechanism in the success of self-management intervention, especially when first attempting new approaches to self-management. A decrease in the IPQ Identity score may reflect a reduction in the number of symptoms the person experienced overall or a re-attribution of symptoms to causes other than RA. Changing symptom perception or re-attributing symptoms to factors other than RA, such as increased activity, may change both emotional responses and coping efforts. These in turn may enable people to engage more actively in self-management behaviours reducing the perceived impact of RA on valued areas of their life and increasing their confidence in their ability to manage pain and other symptoms of RA. People monitor their symptoms and incorporate this information into their decisions regarding self-management behaviour. Therefore, changing the perception and meaning of symptoms could be an important way to influence health outcomes.

Changes in illness representations following self-management intervention could occur because of direct changes in the meaning of symptoms as a result of information and

cognitive restructuring or indirectly by feedback from appraisals of change in symptoms or emotional state resulting from engagement in active attempts to cope with RA. Although the changes in identity and perceptions of control may result from the intervention it is also possible that factors independent of the intervention may have caused the change (e.g. changes in disease activity or mood). Although the precise mechanism of change is unclear, it is clear that the importance of change in illness representations as factors influencing future health outcomes and the direction of the relationships have been demonstrated.

Overall the results of the mediator analyses showed that adaptive changes in illness representations explained the improvements in individualized quality of life and self-efficacy found following self-management intervention. The moderator analyses also suggested that self-efficacy and coping procedures may be important in the maintenance of treatment gains. These results demonstrate, in causal analyses, the role of illness identity, perceptions of controllability, self-efficacy for pain and the use of pain coping strategies in predicting individualized quality of life and self-efficacy in chronic RA. This confirms previous studies, using longitudinal designs, indicating that self-regulatory processes predict (Scharloo *et al.*, 1999) and moderate and mediate (Schiaffino & Revenson, 1992) future health outcomes in chronic RA. Importantly, by experimentally manipulating self-regulatory processes using a self-management intervention, the design of this study significantly strengthens the evidence that self-regulatory processes influence health outcomes in chronic RA.

Some tentative conclusions can be drawn from these results. The moderator analyses aid identification of those people who are most/least likely to benefit from intervention. People who are more confident in their ability to manage pain and try to ignore pain are more likely to maintain improvements in individualized quality of life and self-efficacy for other arthritis symptoms made during self-management intervention.

Second, the mediator analyses increase our understanding of the mechanisms of change through which treatment is effective. The benefits of the self-management intervention,

improved individualized quality of life and self-efficacy, were mediated by changes in the perceived range and frequency of RA symptoms and the perceived controllability of RA. In other words, improvements following self-management intervention were only found if there were adaptive changes in the person's illness representation.

Some suggestions can be made about how interventions may be developed in order to maximise effectiveness of treatment. Interventions could be modified to further enhance self-efficacy and the use of adaptive coping strategies to optimize maintenance of treatment gains. Interventions should also focus on approaches that are likely to reduce the perceived range and frequency of RA symptoms and increase the perceived controllability of RA.

10.5. Methodological considerations and study limitations

Although the results have provided insight into the potential benefits of a self-management intervention and the role of psychological factors in the outcome of chronic RA, there remain a number of methodological inadequacies which limit the conclusions which can be drawn from this study. Methodological considerations will be discussed to provide a context for the discussion of theoretical implications and recommendations for future research and treatment.

10.5.1 Design

The study compares the effects of a self-management intervention plus standard medical care with standard medical care in a single blind randomised controlled trial. This pre-test post-test control group design rules out threats to internal validity (e.g. random assignment rules out selection bias and the control group rules out history and maturation). The design does not rule out pre-test sensitisation. However, the pre-test allowed comparison of groups on response variables before intervention. Significant differences between the groups at baseline were then controlled for in the statistical analyses. A further strength of the study design is that the baseline phase allows detection of

changes in the outcome variables prior to intervention. The intention-to-treat analyses control for the possible influence of selective drop out from the study.

Following Zelen's modified RCT design when participants were recruited they were not initially told they were entering a treatment study. This controls for a potential selection bias, avoiding recruiting only participants who are highly motivated to engage in self-management intervention. Following recruitment and group assignment only participants in the treatment group were told that they were participating in a treatment study, as being involved in a study may be as important as attending an intervention. If participants in the control group knew they were not receiving a potentially beneficial intervention they might feel aggrieved which could affect the extent to which they felt they were usefully contributing to the study. Although ethical approval was obtained for this study, it is likely that if an ethics committee considered the study design now, it would not be approved because of modern research governance guidelines. Failure to disclose the full purpose of the study to the control group participants is now regarded as ethically unacceptable.

Previous studies of the ASMP have used a waiting list control group design, in which the participants in the control group receive the intervention after 4 months. As a result it is not possible to be certain about the long-term effects of the ASMP because the intervention and control group cannot be compared beyond this point. The design used in the present study allowed comparison of the treatment and control groups at 9-month follow-up. A waiting list control group was not used because participants would have had to remain on the waiting list for an unacceptable length of time - more than one year.

10.5.2 Sample size

One limitation of the present study was the sample size. Although 136 people were recruited into the study, after attrition full data were only available for 105 participants (treatment=58 control=47). This limits the power of the study. It should still be sufficient to detect moderate-large effect sizes but small changes may be obscured.

Studies of the ASMP, using mixed diagnostic groups, have tended to have much larger sample sizes. The higher quality RCTs of CBT for RA tend to have smaller sample sizes. Of the CBT studies reviewed by Astin *et al.*, (2002) only Parker *et al.*, (1995) had a larger sample size (n=141).

The sample size may be particularly problematic for detecting treatment effects because 29.9% of participants in the treatment group did not start or complete treatment. However, 47 participants did complete treatment which is still larger than most other studies of CBT for RA.

One possible indication that sample size was not sufficient to detect treatment effects is the finding of sustained improvements in PGI but not in the primary outcome measures. The PGI may be particularly sensitive to change following a self-management intervention as it assesses the specific areas of life that the individual believes have been most affected by their RA. These are likely to be areas that the individual attempts to change during the self-management programme.

Although the sample size is potentially a problem, it can be argued that when considering the administration of a relatively expensive intervention, that small effect sizes would be of questionable clinical relevance. If the effects of self-management were small it would be difficult to argue that they should be provided as part of routine rheumatology services. However, changes which are of both clinical and statistical significance are more compelling.

The small sample size may also be problematic for examining predictors, moderators and mediators. Therefore, the results of these analyses should be interpreted with caution. It is likely that predictors, moderators and mediators were obscured by the lack of power and this makes it difficult to draw definitive conclusions about the mechanisms involved. Therefore, the interpretation of these results can be considered largely hypothesis generating rather than conclusive.

The power of the study could have been increased by recruiting a larger sample. It was not possible to recruit more people with RA from the hospital out-patient clinic, during the recruitment phase the vast majority of eligible patients, 400, were identified and invited to participate. If the recruitment phase had been extended some more patients could have been recruited but this was not possible given the time-limited nature of study funding. Alternatively, patients could have been recruited from a second hospital out-patient clinic but this was again not practical given the resources available for the study. A third possibility would have been to relax the eligibility criteria and recruit people with other types of arthritis. However, the study was designed to investigate the efficacy of self-management for people with chronic RA and the increased heterogeneity of the sample would have compromised the quality of the study. An alternative approach which may have increased the power of the study would have been to increase the numbers in the treatment group starting treatment. If potential participants were told that the study involved attending a treatment programme at recruitment then it is likely that a higher proportion of those agreeing to participate would attend the treatment. However, this could have biased the recruitment towards people who were motivated to attend self-management programmes.

Although the design and inclusion criteria have led to a restriction in the sample size below a desirable level, the power remains sufficient to allow the detection of moderate to large effect sizes. Since the stringent criteria have allowed a more methodologically rigorous experimental design and a more homogeneous sample, it seems likely that the benefits of this approach outweigh the limitations of power in the present study.

10.5.3 Lack of participant blinding and attention placebo control group

Blinding of patients to the treatment condition and the inclusion of a placebo control are generally regarded as important criteria for high quality RCTs. However, both of these conditions frequently cannot be met in psychosocial interventions (Astin *et al.*, 2002). The design of the present study meant that the control group did not know about the

treatment group. However, the treatment group were not blind to the intervention which could have influenced their responses on self-report measures.

In addition, a limitation of the present design was the lack of an attention placebo control group. The treatment group received 12 hours more input than the control group. It cannot be ruled out that it was the non-specifics of the therapeutic relationship which were responsible for the changes which were observed. However, it is important to point out that in those studies that have included an attention placebo group, where changes have been demonstrated this has been primarily in respect of anxiety (e.g. Bradley *et al.*, 1987). Other studies have failed to find significant improvements in either an attention placebo control group (Parker *et al.*, 1989), an occupational therapy control group (Kraaimaat *et al.*, 1995c) or from specific interventions with some cognitive or behavioural strategies (e.g. Shearn & Fireman, 1985; Strauss *et al.*, 1986). Therefore, it would not be predicted that an attention placebo group would have led to the changes observed in the current sample. While it seems unlikely, given the clinical significance of results, particularly for quality of life and self-efficacy, that these results are attributable to non-specific aspects of the intervention, this remains an empirical question.

The use of attention placebo control groups also raises ethical issues and may discourage people from seeking treatment in the future. In addition, although including an attention placebo control group would have been a stronger design experimentally, it would have compounded the limitations of power observed in the present study if a routine medical management group was included as well. Had an attention placebo group been included instead of the standard care group, and no differences between the treatment groups been demonstrated, it would have been difficult to conclude whether or not either treatment was beneficial. This result may have reflected the fact that neither treatment was more effective than standard care. As such, it seemed important to first demonstrate the effectiveness of the self-management intervention prior to comparing it in a larger trial with a non-specific treatment to control for the effects of attention, support and treatment expectancies.

10.5.4 Failure of randomization

In the present study a list of random numbers was generated and patients were allocated on the basis of the order of presentation. Despite this, the groups were significantly different on some measures at baseline. Most importantly, the treatment group had lower tender joint scores than the control group. In addition, the treatment group had significantly more children and were using stretching exercises more. The baseline analyses also show the treatment group is more socially active than the control group, going on more outings and seeing friends more. This is supported by the findings from the ANOVA, that over the study, the treatment group reported significantly greater social activity and support from family (AIMS Social) than the control group. Interestingly, this difference was not significant when initial Ritchie Index scores were controlled for, suggesting that differences in social function may be accounted for by differences in tender joint scores.

Although differences in tender joint score were statistically controlled for in the analyses for Hypothesis 1.1 and 1.2, the failure of randomization remains problematic. There may have been alternative ways in which to have allocated patients to treatment. Treasure & MacRae (1998) have argued that, as in the current trial, randomization does not necessarily guarantee equivalence of groups and yet is still generally considered the gold standard for clinical research. They propose that the method of minimisation may yet become the platinum standard. They argue that “if there are many possible prognostic factors, there will almost certainly be differences between the groups despite use of random allocation” (Treasure & MacRae, 1998). Indeed, in this trial that was the case. They argue that even when the difference is statistically controlled for there is “always an air of uncertainty about the validity of the conclusion”. They recommend a technique called minimisation whereby group allocation is determined by statistically examining the effect of allocating a given participant to any group in a study and allocating the participant on the basis of equivalence between groups. The argument is then that all through the trial allocation, decisions are based upon making all groups in the experimental design as equivalent as possible and hence the likelihood of finding

differences on completion of the trial is reduced. Another alternative which has been suggested is randomised block design, which allocates patients randomly, but controlling for particular variables of interest (Ross, 1998).

The problem with both these methods, and particularly the block method, is which factors would be used as the blocking variable or the variables that determine allocation? In the present study there is a myriad of potentially confounding variables and to have blocked for all of them would have been impossible in a small trial. Indeed, it would be equally difficult to know how minimisation might manage all the variables in the present trial given the limited sample size and use of batch recruitment.

Regardless of the method of allocation, the differences which emerged need to be addressed. Although there is evidence of differences between groups on a few measures overall on a very wide range of other variables the groups were very similar. In addition, the initial scores were used as a covariate to reduce any bias that could affect the results. While it would be difficult to argue that the difference on these variables has affected the entire results of the trial, the finding of a significant increase in stretching exercises, in the whole sample, does need to be interpreted with caution.

10.5.5 Floor effects

Participants in the present study had relatively low levels of depression and anxiety (HADS) compared to those in some other studies. This may have limited the opportunity to demonstrate significant improvements in emotional state. Long-term improvement in depression is one of the most consistently reported findings of psychological treatments for RA (Astin *et al.*, 2002). It was also the only primary health outcome benefit reported by Barlow *et al.*, (2000) in their evaluation of the ASMP.

10.5.6 Assessment of change in individualized quality of life

The procedure for assessing change in individualized quality of life (PGI) was developed specifically for this study. The method was used to ensure that change on the PGI reflected change in the impact of RA on areas of life that the participant had identified as being affected by RA at baseline, rather than reflecting a shift in focus to new areas or priorities. However, the procedure is novel and this might call into question the finding of significant change in PGI following the self-management intervention. Interestingly, a longitudinal study of the PGI in Ankylosing Spondylitis has recently reported successfully using the same procedure to assess change over time (Haywood *et al.*, 2003).

10.5.7 Measurement of illness representations

This study used an early version of the IPQ because it was the only one available at the time. However, subsequent research has suggested some weaknesses with this early version of the IPQ which raise a number of methodological issues (Weinman *et al.*, 1996).

The Identity scale asked participants to rate how often they experienced 12 symptoms as part of their arthritis. From an operational point of view this may well reflect somatization or the tendency to report symptoms, rather than the concept of illness identity, which is the process of matching symptoms to an illness label. A Revised Version of the IPQ, the IPQ-r, has been developed which addresses this and other problems (Moss-Morris *et al.*, 2002). On the IPQ-r people are asked not only to identify what symptoms they have experienced but also whether they believe the symptoms are part of their illness. A newer version of the IPQ, currently in development, also asks people whether they believe any of the symptoms are side effects of their treatment (J. Weinman, personal communication, June 9th, 2003). Therefore, interpretation of the Identity scale used in this study should be cautious.

Internal consistency of some of the IPQ scales was not good, especially the Total Attributions scale and the Control/Cure scale. As Weinman *et al.*, (1996) suggest it may not be appropriate to combine the different cause items into a single scale. However, analysis of

single cause items would have considerably increased the number of variables in the analyses. Therefore, in the present study, cause items were dicotymized and summed to produce a scale measuring the number of causal attributions that people made for their RA. Given the low internal consistency of this scale, findings for the Total Attributions scale should be treated with caution. An alternative approach, which has been used in several studies, would have been to factor analyze the cause items. These studies have identified a number of cause dimensions including biological cause (e.g. immune system, germs and viruses; Heijmans, 1998), emotional cause (e.g. stress and depression; Moss- Morris *et al.*, 1996), environmental cause (e.g. pollution and chemicals; Heijmans, 1998; Heijmans and De Ridder, 1998) and psychological cause (causes such as mental attitude, overwork and personality; Moss-Morris *et al.*, 2002; Rutter & Rutter, 2002).

Another limitation of the early IPQ is that certain items may not be appropriate for a chronic illness group such as RA. The Time Line scale contains items assessing perceptions about whether RA is acute or chronic and the cause items assess perceptions about what has caused RA. In people with a confirmed diagnosis expectations about whether specific aspects of the disease will change over time may be more salient than beliefs about whether the disease is acute or chronic. In a study of people with early RA those who believed their pain would continue (chronic time line) reported more depressive symptoms two years later compared to those who thought their pain would go away or come and go Skevington (1993). A study in chronic RA has shown that perceived causes for RA are not related to health outcome but that perceived causes for symptom flares and remissions are (Affleck *et al.*, 1987a).

Studies of the early version of the IPQ have shown that the internal consistency of the timeline and control/cure sub scales is problematic (Weinman *et al.*, 1996). Weinman *et al.*, (1996) re-analysed the control/cure sub scale and found that it is measuring two independent factors 1. Personal control and self-efficacy, and 2. Beliefs about the efficacy of treatment. The IPQ-r separates these different aspects of control/cure representations and similarly separates "acute vs chronic" from "persistent vs cyclical" timeline perceptions (Moss-Morris

et al., 2002). Given this, findings in relation to control/cure and timeline in the present study should also be treated with caution.

Although there are problems with the early version of the IPQ, used in this study, there are ways in which some of the problems with the measure might have been addressed. Scharloo *et al.*, (1999) have reported that interviewing people about their illness representations prior to completing the IPQ makes it easier for them to complete, a "priming" effect. Scharloo *et al* 1999 also combined data from interviews with IPQ data to improve the internal consistency of the emotional attributions and consequences scales. In addition they selected subsets of items from the Cause and Control/Cure scales as these items had particular salience for RA patients. This was not done in the present study as it was felt that it would be important not to exclude potentially significant aspects of people's illness representations. However such an analysis of selected subsets of items might have been useful.

An important caveat here is that Leventhal *et al.*, (1980) suggest that individuals will exhibit a characteristic illness representation profile for each illness according to its symptomatic features and chronicity. It is for this reason that some researchers (e. g. Turk *et al.*, 1986; Heijmans, 1999) have argued in favour of factor analysing the theoretically derived items from instruments designed to measure illness cognition. This would identify the parsimonious categories about which people cluster their lay- views regarding their illness. However, it can be argued that since the theoretically derived dimensions originated from extensive pilot work (Leventhal *et al.*, 1980; Weinman *et al.*, 1996) and the factor analyses usually extract factors that do not deviate greatly from these dimensions, the use of the theoretically derived dimensions is a productive and fruitful endeavour. Further, the theoretically derived Dimensional structure has been adopted by the majority of quantitative investigations into illness representations. The five dimensions of cause, consequences, cure/ control, identity and timeline are recognised as the basic building blocks of inquiry into how individuals construct a representation of their illness (Heijmans and de Ridder, 1998).

10.5.8 Multiple comparisons

The number of statistical tests conducted on the same data set increases the risk of a type 1 error. In some analyses a Bonferroni correction was made. However, given the exploratory nature of some of the analyses this was not always appropriate. Therefore, some of the findings must be treated with caution.

An alternative approach might have been to use composite scores for some of the variables. For example, research has supported the reliability and validity of a composite Arthritis Self-efficacy Scale score (total self-efficacy), based on a sum of the 3 ASE scale scores (Parker *et al.*, 1995). However, as previous research has shown that only some of the ASE scales change following self-management intervention (Barlow *et al.*, 2000) it was decided to analyse all the scales separately. Nicholas, Wilson and Goyen (1991) combined the scales of the Coping Strategies Questionnaire into a single active coping measure, unfortunately this could not be used in the present study as a modified version of the CSQ was used which omitted some of the CSQ scales. It might also have been useful to use a disease activity index as has been used in some other studies of RA (Scharloo *et al.*, 1999; Young *et al.*, 2000) rather than specific measures of disease activity.

10.5.9 Direction of causation

The longitudinal analyses testing Hypothesis 2.4 support the causal assumptions underlying the SRM but do not rule out other causal explanations. By controlling for socio-demographic factors, baseline disease activity, pain, physical disability, emotional distress and social function the probability of this explanation is minimised. However, it is possible that change in a third variable, not included in the analysis, could explain both the changes in self-regulatory variables and changes in health outcomes. For example, decreases in the perceived range and frequency of RA symptoms, and increases in confidence to manage pain and perform functional activities explained changes in disability. However, decreases in disease activity could have caused both of these

changes. An alternative approach would have been to enter concurrent disease activity in the analysis. This was done in the Scharloo *et al* 1999 study but the composite disease activity/severity measure did not explain a significant amount of variance in health outcomes. Furthermore, in the present study, the baseline cross-sectional analyses revealed that even controlling for socio-demographic factors, disease activity, physical and social function the self-regulatory variables were the strongest predictors of health outcomes in many of the analyses.

More research is needed to disentangle the complex bi-directional relationships between self-regulatory processes and health outcomes suggested by the SRM. Intervention studies, directed at influencing specific self-regulatory processes that have theoretically predictable relationships to objective health outcomes, could be used to explore these mechanisms.

10.5.10 Comprehensive self-management intervention

The use of a multi-component treatment makes it difficult to identify what the active treatment components are. It may be that had the intervention focused more on some skills and less on others that the treatment response of participants may have been facilitated or alternatively lessened. While this is a theoretical weakness of the present study, it is likely to be an empirical strength. There is evidence to suggest that broad-based interventions are more effective than single component treatments (Young, 1992). It is possible that it is a combination of the methods used, across the treatment programme, which contributes to the effects observed. Alternatively, it is possible that different participants take different skills from the programme and hence multi-component treatments allow participants to identify those strategies which are most useful to their situation. As such, limiting the programme would potentially limit either the efficacy across participants or the proportion of participants for whom the programme was useful. Post-intervention interviews or focus groups with participants could have been used to explore what aspects of the intervention participants believe were most helpful. The same team of health professionals led all the self-management programmes and they followed a detailed course leaders' manual. However, it

is possible that over the course of the study subtle changes in the way the intervention was delivered could have developed. Independent verification that the self-management programme was being delivered according to the protocol would have been useful. These and other difficulties of conducting trials of complex interventions have been recognised by the Medical Research Council and are discussed in their recent guidelines (MRC, 2000).

10.5.11 Maintenance of treatment gains

The present study has demonstrated that for both quality of life and self-efficacy for pain and other arthritis symptoms gains were maintained over the 9-month follow-up period. Nonetheless, in the context of a chronic disease, 9 months is a very short period from which to argue maintenance of gains. It remains an empirical question as to whether these gains will result in longer-term benefits to patients.

10.5.12 Generalisation of findings

The inclusion and exclusion criteria limit the extent to which the results can be generalised. Participants in this study were drawn from a hospital outpatient rheumatology clinic. Participants had to be aged 16-70 and have a confirmed diagnosis of RA of at least one-year duration. Therefore, no firm conclusions can be drawn about the generalisation of results to people with RA who are not attending hospital out-patient clinics, children or older people with RA or those with early RA.

There was a reasonable rate of uptake in the present study for those approached (34%). Participants were representative of people with RA attending the out-patient clinic who were eligible to participate. The only difference between participants and those who declined to participate was that participants lived nearer the research centre. These factors are likely to increase the extent to which findings can be generalised. No other studies of psycho-educational intervention for rheumatic disease could be identified that reported findings comparing the sample to the population eligible to participate.

The drop-out rate in the present study is not high and is impressive given the demanding nature of the study (five assessments over one year, requiring completion of questionnaires, visits to the hospital, blood tests and for the treatment group attending six group sessions). Of 136 people who consented to participate in the study, when first approached, 18 (13.2%) provided no data. This is a response rate of 86.8%. Of the 118 participants who provided data 13 (11%) withdrew from the study. Overall, 22.8% provided no data or withdrew. Studies differ in their definitions of "drop outs" but these figures are in line with those reported in similar intervention studies (e.g. 17.3%; Taal *et al.*, 1993b) and longitudinal studies (e.g. 24.5%; Scharloo *et al.*, 1999).

In addition, there were few differences between participants who completed the study and those who provided no data or withdrew. The 18 participants who provided no data were significantly older and had had RA longer than the 118 participants who provided data. The 31 participants who provided no data or withdrew from the study were significantly older than the 105 participants that completed the study. It is possible that self-selection among participants might have biased the sample towards slightly younger people. This might have influenced the results to some extent. Some studies have shown that age is associated with health outcomes in RA (e.g. pain Parker *et al.*, 1988a; HAQ Anderson *et al.*, 1988; depression Wright *et al.*, 1998). In addition, Parker *et al.*, (1988b) suggested that older people with RA might benefit less from psycho-educational interventions. However, as the difference is small it is unlikely to have had a major effect on the results. The 13 participants who withdrew from the study had significantly lower Self-Efficacy for Pain. It is possible that treatment could be less effective for older people or those with less confidence in their ability to manage pain. However, the intention-to-treat analysis controlled for the selective effects of withdrawal.

Stoke Mandeville Hospital, from which the current sample was recruited, provides services to people living in central Buckinghamshire and west Bedfordshire. This is a broad catchment area with several large towns and rural areas. The catchment includes areas of relative affluence and areas with less fortunate economic circumstances. It seems

likely that the participants in this study would be reasonably representative of people with RA attending hospital out-patient clinics throughout the UK.

However, it is noteworthy that the ethnic origin for all participants was "white UK" or "white European". This may reflect the underutilization of medical services in the UK by patients from different ethnic groups. However, as a result, it is unclear how these results might generalise to other ethnic groups where the meaning of illness might differ significantly.

Notwithstanding this caveat, the high up-take rate, low drop-out rate and range of sociodemographic experiences of patients from the catchment area would all support the generalizability of results to UK hospital out-patients with chronic RA.

The success of an intervention such as this with a group of people who were not selected on the basis of having difficulty in managing their RA or being motivated to engage in a self-management intervention is a more stringent test of the intervention than previous evaluations of the ASMP.

10.5.13 Possible adverse treatment effects

It is important to consider the possibility that, while the treatment in the present study had benefits across the sample investigated, some individuals within that sample may have responded adversely. There is certainly no evidence to support the fact that patients became significantly worse over the course of the intervention.

All participants who started treatment, including the 6 participants who did not complete treatment (attending 1-3 sessions), completed post treatment assessments at time 3. No adverse effects of treatment were reported suggesting that the treatment does not cause any immediate problems. However, 3 participants who attended 1-3 treatment sessions and two who attended 4-6 sessions, withdrew from the study during the follow-up phase.

Therefore, it is not possible to be completely certain that the treatment had no long-term adverse effects.

There were few differences between those who completed treatment and those who did not. A significantly higher proportion of participants withdrew from the study in the group that did not complete treatment than in the group that completed treatment. Analysis of the timing of withdrawals reveals that they did not withdraw from the study because of the treatment but rather were unable to start treatment because they had already withdrawn from the study or they withdrew during the follow-up phase.

The group who completed treatment had a significantly higher mean score for frequency of performance of household tasks at the start of the study. It is possible that people who have more difficulty in performing domestic activities may not successfully engage in self-management intervention. An alternative explanation is that those people who spend less time on domestic activities may be working or have other demands on their time, which in turn may make them less likely to complete the self-management programme.

10.5.14 Ecological validity and cost-effectiveness

People with RA rarely have access, at least in the UK, to psychological services. Where psychological services are available it is typically reserved for patients identified by Rheumatologists as being psychologically vulnerable in some way. This can change the nature of the relationship between therapist and patient. Skevington (1986) has argued that patients who feel that their diagnosis is not validated may well behave in a variety of ways to legitimise their illness. She has argued that this may be the case with seronegative patients. However, equally patients referred to Psychology Services, especially where these are housed within an Adult Mental Health Department, may believe that their RA is not taken seriously by their rheumatology team. One can assume that engaging in psychological therapy is unlikely to be a method by which patients would legitimise their illness and hence there may be difficulties inherent in clinical practise while Clinical Psychologists remain working outside of Rheumatology Clinics.

In the current economic climate of the National Health Service, it would be difficult to imagine comprehensive provision of Psychology Services, working within Rheumatology Clinics, routinely seeing patients. The present study, however, would suggest that there may be health benefits associated with increased provision of psycho-educational interventions. To achieve this, it would be necessary to provide a strong argument, not only in terms of improved quality of life, but also economic benefits. Cost-effectiveness may even prove to be a stronger argument than improved quality of life. Although psycho-educational interventions for people with RA may only produce modest changes in health status they have been shown to have a very significant impact on long-term health-care costs (Lorig *et al.*, 1993; Young *et al.*, 1995). It is a gap in the current study that the only measure of health-care utilization included was visits to doctors. A full analysis of the socio-economic benefits of self-management intervention for people with RA would inform the development of future health policy and provision (Clarke, 1997; Groessl & Cronan, 2000).

10.6 Theoretical implications

The findings provide support both for the importance of psychological factors in RA and the particular mechanisms which may be operating. The present study was guided by Leventhal's SRM but there are other theories of health behaviour, which could be helpful in attempting to understand the above findings. The implications of the findings for other theories will be discussed first followed by a discussion of the implications of the findings for the SRM. Future research could allow designs which could explicitly test the differences between models in the context of the current findings.

10.6.1 Self-efficacy and Social Learning Theory

Self-efficacy has been identified as an important factor in adaptation to rheumatic disease (Marks, 2001). Bandura (1977) described self-efficacy as an important mediator of motivation to perform specific behaviours within his Social Learning Theory (SLT). In addition, self-efficacy has also been incorporated within other social cognition models,

for example the Theory of Planned Behaviour (TPB) and Protection Motivation Theory (PMT). Bandura's SLT has been widely applied in psychological research on arthritis and was influential in the development of the ASMP.

The results provide some support for the importance of self-efficacy in adaptation to chronic RA. First, in relation to the results of the analyses of treatment efficacy, Rhee *et al.* (2000) suggest that the reason why previous psychological interventions have failed to find significant changes in health outcomes is because they have not changed self-efficacy. Consistent with this, in the present study, there were significant changes in both self-efficacy for pain and other arthritis symptoms and changes in pain and depression immediately following the intervention. However, Bandura's SLT, and other theories such as the TPB, may have difficulty explaining the long-term results. It is difficult to understand why, although people remained confident in their ability to manage pain and other arthritis symptoms at follow-up, the improvements in pain and depression were not maintained. This suggests that while changes in self-efficacy may be a necessary condition for improvement in some health outcomes they are not always sufficient to produce sustained improvements.

The SRM is a useful template for exploring why interventions may not be successful. One obvious problem with both self-efficacy theory, and the ASMP which was based on it, is a lack of attention to emotional regulation and appraisal processes. As discussed earlier, the self-management programme may enable people with RA to become more confident in their ability to manage their RA and to adopt new self-management behaviours which lead to improved health outcomes. However, when faced, in the future, with episodes of uncontrollable disease activity, increased pain and disability they may appraise their efforts to achieve their goals more negatively. The SLT would suggest that this could lead to a reduction in self-efficacy which in turn would influence performance of health behaviours and health outcomes. However, in the present study there was no reduction in self-efficacy but health outcomes did decline. The self-regulatory processes proposed by the SRM could explain these results. Feedback from appraisals of coping could lead to increased emotional distress, a more negative illness representation and

subsequently more maladaptive coping and poorer health outcomes, creating a vicious cycle. The failure to maintain, at follow-up, the adaptive changes in illness representations, coping strategies, mood and pain found immediately after intervention, despite sustained improvements in self-efficacy, would support this proposition.

The cross-sectional and longitudinal analyses also provide some evidence for the importance of self-efficacy. Both baseline and change in specific self-efficacy measures were found to explain unique variance in a number of health outcomes. Although, as discussed earlier, at least part of these associations may be due to measurement artefact because of the overlap between self-report measures of pain, disability and emotional distress and the self-efficacy scales. Of course, it is also possible that a third factor could explain these relationships.

Another important issue is whether the Arthritis Self-efficacy Scale only measures self-efficacy. As discussed earlier, many of the items seem to be tapping both self-efficacy beliefs and outcome expectancies e.g. "How certain are you that you can decrease your pain quite a bit?" and "; "How certain are you that you can control your fatigue?". Furthermore, it is interesting that Rhee *et al.*, 2000 found that it was a composite measure, including the ASE, AHI and the PCRT factor of the CSQ that mediated improvements in pain and depression. These other measures also include items assessing beliefs about one's ability to control symptoms and negative outcome expectancies. It remains unclear whether it is the belief that one can perform specific symptom management behaviours, the belief that one can control the level of symptoms or a combination of these that is the most important predictor of future health outcomes.

The moderator and mediator analyses could potentially provide stronger support for the role of self-efficacy. In the moderator analysis those who had lower confidence in their ability to manage pain, at baseline, showed less improvement in individualized quality of life following the intervention. Within Bandura's SLT An interpretation of this finding would be that those who lack confidence in managing pain are less likely to adopt new self-management behaviours and subsequently experience less improvement in quality of

life. Alternatively, even if they do adopt new behaviours during the intervention they may stop them following the intervention because they appraise their ability to cope with RA more negatively. This second explanation is more likely as most people greatly increased their use of a range of self-management behaviours during the programme but there was some decline during the follow-up period, and self-efficacy only moderated quality of life at the 9-month follow-up. It is also possible that self-efficacy might influence quality of life through cognitive rather than behavioural mechanisms. Lorig & Gonzalez (1992) report that although changes in self-efficacy were strongly associated with the outcome of the ASMP changes in self-management behaviours were not. This suggests that improvements in health outcomes could have been mediated by changes in self-efficacy not behaviour change. To speculate people who are more confident in their ability to successfully perform behaviours to manage their RA may perceive that RA has less impact on valued areas of their life.

Sharpe (1999) has observed that, while the concept of self-efficacy is useful, there are difficulties in interpreting the results of self-efficacy studies because of a conceptual confusion in the self-efficacy literature. If an individual has a low level of self-efficacy for a particular behaviour, this can mean one of three things. First it may mean that the individual is not very competent at that particular behaviour and they are accurately appraising their low level of ability. Alternatively, they can be very skilled at a particular behaviour, but have no confidence, and as such appraise themselves negatively. Finally, it is also possible that an individual may have a relatively low level of competence, but also appraise themselves more negatively than is accurate. The inability of the concept of self-efficacy to distinguish between these alternative interpretations is problematic.

This raises the possibility of an alternative interpretation of the moderator analyses. It is possible that those who have more difficulty managing their pain are accurately evaluating their ability. Despite increases in their confidence to manage their symptoms, and the use of new approaches to self-management, As their pain is less well controlled they are less able to maintain gains in quality of life.

The findings of the present study suggest that confidence in one's ability to manage the symptoms of RA is important for successful adaptation. However, there is less support for Bandura's SLT or the TPB. First, the present study found that in addition to self-efficacy, other self-regulatory variables not included in these theories (e.g. illness representations) were important predictors of health outcomes. Second, in the present study although the intervention produced significant changes in self-efficacy, they did not mediate the improvements in individualized quality of life, but changes in illness identity did. Third, in an illness like RA, where levels of disease activity, pain and disability can fluctuate dramatically, it is difficult to see how the relatively static processes proposed in the SLT and TPB can provide an adequate description of the complex dynamic processes of self-regulation that occur over time (Keefe *et al.*, 2002). Finally, these theories neglect the role of important processes, such as emotional self-regulation and appraisal.

Recent descriptions of Leventhal's SRM (Cameron & Leventhal, 2003; Leventhal, Brissette, & Leventhal, 2003) and other self-regulatory models (Scheier & Carver, 2003) have incorporated self-efficacy or confidence within their self-regulatory frameworks. Leventhal's SRM focuses less explicitly on beliefs about one's ability to perform a specific behaviour, although beliefs about disease controllability and expectations of treatment efficacy are seen as implicitly embedded within illness and treatment representations (Cameron & Leventhal, 2003; Leventhal *et al.*, 2003). Cameron and Leventhal (2003) suggest that self-efficacy and competence beliefs may be crucial in the adoption of complex health behaviours (e.g. exercise) yet less important for a substantial proportion of other illness behaviours. For example, there may be little variation in perceptions of one's ability to take oral medication or seek medical care for serious symptoms. Overall, self-efficacy seems to be important in adaptation to RA but placing it within a broader theoretical framework may lead to a better understanding of its role in the complex dynamic processes of self-regulation.

10.6.2 Generic self-regulatory models

A key factor that distinguishes between different self-regulatory models used in health psychology is whether they represent a general model of behaviour (e.g. the Carver and Scheier model or the Lazarus and Folkman stress and coping model) or whether it applies specifically to health and illness behaviour (e.g. Leventhal's SRM). General models are useful for integrating work on self-regulation across different psychological domains whereas health specific models may capture elements of health and illness that are unique to this domain. These theories operate at different levels of specificity and advances at one level may inform and shape understanding at the other level. Greater attention to the fit between general and specific models will ensure that theoretical developments at both levels are consistent.

The SRM and the Carver and Scheier models complement each other. The SRM has been developed primarily to explain health and illness behaviour and pays particular attention to schematic content. The Carver and Scheier model has developed as a more general theory of behaviour and elaborates on the process of self-regulation. General models have provided a number of valuable insights into the process of self-regulation of health and illness. For example, Carver and Scheier (1996) suggested that people have two overarching goals of survival and coherence. Illness experiences threaten both survival and a sense of coherence in ones sense of self and life goals. This emphasizes the critical importance of illness related events and why adaptation to illness can present critical challenges in self-regulation. In RA both survival and coherence are likely to be critical aspects of representations and goals.

10.6.3 Implications of the findings for the SRM

It may be argued that the SRM is not a "true" theory, as the breadth, complexity and dynamic nature of the SRM makes operationalising testable hypotheses difficult. The SRM has generally been used as a framework for guiding research, highlighting variables and processes that may be important within a specific situation. However, some of the

assumptions within the SRM have been examined in empirical studies using a variety of chronic illnesses (Kaptein *et al.*, 2003; Hagger & Orbell, 2003). These studies have provided evidence for the construct and discriminant validity of the dimensions of the cognitive representation of illness proposed by the SRM. Further, theoretically predictable relationships between illness cognitions, coping and outcomes have been identified across studies (Hagger & Orbell, 2003).

For example, the Leventhal *et al.*, (1980) model makes an explicit link between illness representations cognitions and coping behaviours. The model proposes that the illness representation acts as a filter and interpretive schema for the available sources of information about an illness and guides action in response to the illness threat. Further, the model implies that the relationship is causal, that is, the illness cognition will exact an effect on coping behaviours in proportion with the perceived severity of the illness based on the representation derived from the stimuli.

The findings of the present study are consistent with this proposition. As discussed earlier, perceiving the symptoms of RA to be more frequent and incoherent and the consequences of RA to be more serious were associated with greater use of maladaptive coping strategies and less use of adaptive strategies. Further, perceiving RA to be more controllable was associated with greater use of adaptive strategies. However, perceptions of cause and timeline were not associated with coping strategies in this study. This may reflect methodological weaknesses in the way these concepts have been operationalised in the early version of the IPQ, as discussed earlier, but few other studies of chronic illness have found consistent relationships between these dimensions and coping (Hagger & Orbell, 2003). It is possible that more specific cause or timeline perceptions (e.g. about the cause of a symptom flare or whether symptoms are likely to be persistent or cyclical) may have a greater influence on coping and outcome in chronic illness (Affleck *et al.*, 1987a; Skevington, 1993).

These findings, in conjunction with consistent findings from a wide range of other chronic illnesses (Hagger & Orbell, 2003) provide strong support for the close

relationship between illness representations and coping procedures proposed by the SRM. However, much of the evidence is cross-sectional and further work is needed to test the causal direction of the relationship.

A second key assumption underlying the SRM is that self-regulatory processes mediate between the disease and health outcomes. The relationships between self-regulatory variables and health outcomes were examined in the cross-sectional and longitudinal analyses. The results were in line with the study hypotheses, that self-regulatory variables would explain a significant amount of variance in health outcomes. The results are also largely consistent with studies of other chronic illness (Hagger & Orbell, 2003; Kaptein *et al.*, 2003). Although moderating and mediating relationships were not examined in the longitudinal analyses, these have been examined in some other studies of rheumatic disease (e.g. Hampson Glasgow & Zeiss 1994; Schiaffino & Revenson, 1992). Finally, the finding that decreases in illness identity mediated improvements in individualized quality of life in the treatment group provides strong support for the causal direction of the relationship between self-regulatory processes and health outcomes proposed by the SRM. Overall these findings are consistent with the assumption in the SRM that the disease influences health outcomes via self-regulatory processes.

A major tenet of the SRM is that a causal relationship exists between illness cognitions and outcomes that is mediated by coping. This premise suggests that the negative influence of illness cognitions such as identity on adaptive outcomes like psychological well-being can only be understood through the influence of a relevant coping strategy that may alleviate illness symptoms such as problem- focused coping.

This mediational hypothesis was not formally tested in the present study, and it would probably have been difficult to do this as there were relatively few longitudinal analyses in which health outcomes were explained by both illness representations and coping procedures.

However, in both the cross-sectional analyses and the longitudinal analyses, examining changes in self-regulatory predictors, illness representations were found to explain greater amounts of variance in more of the health outcomes than coping procedures. In the longitudinal analyses, examining baseline self-regulatory predictors, although few self-regulatory predictors were identified, coping strategies and self-management behaviours were found to predict health outcomes more often than illness representations.

There are few studies that have investigated the mediation hypothesis prospectively (Hagger & Orbell, 2003; Kaptein *et al.*, 2003). Several studies have attempted to examine the mediation hypothesis using cross-sectional data but these have had limited success. Studies on CFS (Moss-Morris *et al.*, 1996) epilepsy (Kemp *et al.*, 1999), chronic obstructive pulmonary disease, psoriasis and rheumatoid arthritis (Scharloo *et al.*, 1998) and Addison's disease (Heijmans, 1998) have all reported null findings for the mediation hypothesis. These studies found a significant impact of illness representations on health outcomes with zero or very small additive impacts of coping behaviours.

Hagger and Orbell (2003) have suggested that the weak relationships between coping behaviours and health outcomes and the stronger relationships between illness representation and health outcome may be explained, in part, by the assessment of coping and the possible confounding feedback effects of outcomes on illness representations. Coping measured by questionnaires is limited due to their lack of specificity (Coyne & Racioppo, 2000). One means to counter these limitations is to use more objective, problem-focused behavioural coping measures such as practice of self-management behaviours, compliance with medication or attendance for routine blood tests. These provide illness-specific assessments of coping that are not limited due to their generality. However, such measures may also be subject to bias if the direction or focus of the coping behaviour is not known. For example, doctor visits may be indicative of a coping response related to the target illness, but since actual reasons for the visits are typically unspecified, the researcher cannot be sure that such a response can be definitively and directly attributed to the target illness. Further, in the SRM, feedback from the appraisal of health outcomes can modify illness and such re-evaluations may interfere with

assessing the proposed mediation relationship between illness representations, coping and health outcome. It may be that the strong relationships between illness representations and outcomes are due to a causal relationship between health outcomes and illness representations that could be modelled at the appraisal stage. However, there has been little research either exploring the nature of appraisal processes or developing ways to measure them.

An alternative hypothesis that has not received much attention in the literature is that the influence of cognition on outcomes may be exacerbated or hindered by the coping behaviours. This suggests that a moderating effect exists such that the effect of illness cognitions, such as controllability on illness outcomes like vitality and psychological well-being, may be more influential in the presence of coping behaviours that address controllability such as problem-focused coping.

Future research will therefore need to focus not only on longitudinal designs, but also on formal tests of the SRM to examine whether coping mediates, or moderates, the influence of illness cognitions on illness outcomes.

A number of key features of the SRM were not addressed in this study. Emotional responses are crucial elements of the self-regulatory system (Cameron & Leventhal, 2003). The present study has investigated emotional responses as experiences to be regulated, but they also have an important role as influences on cognition and behaviour and as direct responses to appraisals of goal related progress (Cameron & Leventhal, 2003). In addition, future investigations need to take the appraisal stage of the SRM into account to fully evaluate the complex manner in which illness representations affect coping and illness outcomes.

Specific self-regulatory processes may not be the most important determinants of change for psychological intervention. The integration and coherence of all components in the self-regulatory system may be a more important factor in effective self-management and emotional adjustment (Leventhal *et al.* 1992a). Given that coherence is a primary goal

motivating human behaviour (Carver & Scheier, 1996) this issue deserves further exploration. Leventhal *et al.* (1992a) identify issues of coherence in three domains.

- 1) Coherence of the problem space: consistency between attributes of the illness representation with the coping procedures and the match between the appraisals of coping effects with the outcome expectations suggested by the illness representation. Behaviours regulated by a coherent system can become relatively autonomous and be maintained over long periods of time. Unpredictable symptom flares or side effects of treatment can seriously undermine coherence, given the tendency to interpret symptoms as indicators of disease. Developing a self-regulatory system that is able to integrate the unpredictable and uncontrollable aspects of RA may be a key factor in successful long-term outcome.
- 2) Coherence at the cultural and interpersonal levels: many features of the individual's illness and treatment representations, coping procedures and appraisal are shared rather than private (Weinman, Heijmans & Figueiras, 2003). When coherence is lacking, because individuals neither share nor negotiate common outlooks, optimal self-management may be difficult to achieve. For example, adherence to treatment appears to be high when the patient, health professionals and significant others have common representations, agree upon treatment procedures and share criteria for outcome appraisals (Leventhal *et al.*, 1992a). A group intervention involving the sharing of illness representations, coping procedures and criteria for appraising outcome with peers and health professionals may provide an excellent opportunity to develop coherence between the person and significant others. Interestingly, participants in self-management interventions report that the opportunity to share experiences is a highly valued aspect of the ASMP) (Barlow *et al.*, 1997a).
- 3) Coherence of the problem space and person: Research guided by the SRM has generally tended to focus on the psychological processes that are specific to illness experience but there is increased recognition of the need to connect these self-regulatory processes with the more general self-system (Contrada and Ashmore 1999). Illness challenges the integrity of the self and managing illness requires the regulation of critical aspects of the self (e.g. emotional and physical states). The personal meaning of health related goals influences coping behaviour and appraisals of health status and health related activities shape the self-concept. There is also important work showing that

motivation to protect the self can bias health related cognitions with important emotional and behavioural consequences (Wiebe and Corbell, 2003). A coherent and stable approach to managing RA is unlikely to exist when the self-regulation of the illness is inconsistent with the self-system and this issue needs further investigation.

It is perhaps fair to say that empirical examination of the complex processes proposed in self-regulatory models is at an early stage. Testable predictions are beginning to be developed and the early results are encouraging.

10.7 Future research and treatment implications

There are a number of areas for future research and implications for treatment suggested by the present study and wider literature.

10.7.1 Who is likely to benefit from intervention?

Given the number of studies of psycho-educational intervention for people with rheumatic disease there is remarkably little evidence about who is most/least likely to benefit. Further work is needed to investigate what demographic, physical, psychological or socio-cultural factors (e.g. gender; diagnosis; motivation to engage in self-management) influence response to treatment. For example, the results of the present study suggest that those who are less confident in their ability to manage pain, and who do not try to cope with pain by ignoring it, are less likely to maintain the benefits of treatment. A better understanding of these issues would assist in the selection of people who are most likely to benefit from particular interventions. In addition, it would inform the design of improved interventions to meet the needs of those who are less likely to benefit from existing approaches.

10.7.2 When is intervention likely to be effective?

The present study and wider research literature show that psycho-educational interventions for RA can increase people's understanding of their illness and facilitate the development of adaptive illness-related cognitions and coping behaviours. They have also been shown to improve health outcomes but long-term benefits have not been demonstrated consistently. However, there is evidence that psycho-educational interventions are less effective for those who have longer disease duration or more severe disease (Astin *et al.*, 2002). Sharpe *et al.*, (2001c) has shown in a small-scale study that a CBT group for early RA had some long-term benefits. Larger studies are needed to replicate this finding before recommending that people with early RA should be offered psychological treatment as part of their routine care. Many people are living with chronic RA and the long-term benefits of early intervention are uncertain. Therefore, given the relative lack of short-term and long-term effectiveness of interventions for chronic RA further research is also needed to develop and evaluate effective interventions for people with chronic RA.

At the time when this study was conducted people with RA trained to deliver the ASMP were not available in the UK. With the development of the NHS Expert Patient Programme (Department of Health, 2001) self-management programmes for people with chronic illness (Lorig *et al.*, 1999) are now becoming widely available in the UK. As discussed earlier there is evidence that people who report they have RA, recruited from the community and who are motivated to attend a self-management programme do as well as other people with arthritis attending the ASMP (Barlow *et al.*, 2000). However, given the disappointing long-term results of CBT interventions for hospital out-patients with chronic RA it is possible that this group may not benefit from attending chronic disease self-management programmes. It is possible that programmes led by lay people with chronic illness are more empowering and successful than programmes led by health professionals. Alternatively, as argued earlier, the differences between the results of CBT and ASMP studies could be explained by differences in study design or the populations studied. It remains an empirical question whether hospital out-patients with RA would benefit from attending a self-management programme if it were

led by trained lay people with chronic illness. This question has important clinical, ethical and economic implications and should be investigated before these programmes can be recommended for hospital out-patients with chronic RA. For patients, it is important that they know whether or not a treatment is likely to be effective for them. For health professionals, it is important to weigh up whether participating in this form of treatment is the best approach for their patient. Other treatments may be more appropriate or cost-effective.

10.7.3 What mechanisms facilitate successful adaptation and response to treatment?

There is now considerable evidence about the patterns of illness representations that are associated with maladaptive coping and poorer health outcomes in chronic illness (Hagger & Orbell, 2003; Kaptein *et al.*, 2003). The SRM proposes that changing these illness representations should in turn lead to more effective coping and better outcome. A recent review provides encouraging evidence that psychological interventions that change self-regulatory processes can produce changes in health outcomes for people with chronic illness (Petrie, Broadbent & Meechan, 2003). However, very few interventions have been developed explicitly using the SRM (Petrie *et al.*, 2003). Interventions based more closely on an understanding of how self-regulatory processes influence health outcomes may improve treatment efficacy and provide an opportunity to test the assumptions of the SRM.

The results of the longitudinal study indicate that it is possible to predict those who are most likely to continue to experience high levels of pain, physical disability, emotional distress and poorer quality of life up to a year later. Given this it may be desirable to target scarce psychological resources to those patients most at risk. The study also identified a number of specific self-regulatory variables which seem to be particularly important in predicting health outcomes (illness identity, consequences, self-efficacy and passive coping). Previous interventions have focused on changing self-efficacy and coping but have not attempted to change illness representations. Interventions could be

developed specifically to modify symptom perception and the perceived consequences of RA. It may also be possible to develop interventions tailored to address specific unhelpful self-regulatory patterns.

Self-management and cognitive-behavioural group interventions for RA typically deliver the same treatment package to all participants. An alternative more individualized approach might allow specific maladaptive aspects of people's illness representation, coping and appraisal to be addressed more effectively. Petrie, Cameron, Ellis, Buick, and Weinman (2002) have described an intervention developed using Leventhal's SRM for people who have recently had a myocardial infarction (MI). This intervention was explicitly developed within the framework of Leventhal's SRM to change patients' inaccurate and negative illness perceptions of their MI. This programme used an individualized approach in which the content of each patient's intervention was based on an assessment of their perceptions of their MI. There is extensive evidence that cognitive, emotional and behavioural processes are amenable to intervention with positive effects on outcome. The development of interventions more clearly based on the theoretical and empirical literature might improve treatment outcome further.

The present study used the self-management intervention as an experimental manipulation to examine the influence of change in self-regulatory variables on subsequent health outcomes. This did provide some evidence that changes in health outcomes were moderated and mediated by self-regulatory variables which could be targeted in future interventions. Unfortunately, the changes in health outcomes were modest so this only provided a limited test of the SRM. More powerful manipulations (e.g. drug treatment or surgery) could be investigated to examine whether self-regulatory processes mediate between the disease and health outcomes. Many health outcomes in RA are relatively stable over the medium term. An alternative method for investigating the relationships between self-regulatory processes and health outcomes would be to investigate whether the impact of naturally occurring short-term fluctuations in disease activity (exacerbation and remission) on health outcomes are mediated by self-regulatory processes.

10.7.4 Which components of treatment are necessary?

The present study and wider research has provided potentially useful targets for psychological intervention in chronic RA and other chronic illnesses. For example, Cameron & Leventhal (2003) have noted that effective interventions will facilitate adaptive changes in people's illness and treatment representations, develop new coping strategies and enhance self-monitoring and appraisal of progress. Although this is useful, further work is needed to identify the best methods for facilitating and maintaining change in these self-regulatory processes. Within the field of cognitive-behaviour therapy there is an extensive literature on effective methods for changing cognitions, emotions and behaviours (White, 2000). It is less clear which of these methods are most effective in changing self-regulatory processes or health outcomes for people with chronic physical illness. Most of the interventions for RA have focussed on the use of methods to change beliefs and coping strategies rather than emotional or appraisal processes. There is evidence to suggest that when faced with a chronic uncontrollable illness emotional self-regulation may be particularly important for successful adaptation (Hagger & Orbell, 2003). CBT methods, which have been demonstrated to improve emotional well being, need to be evaluated further in interventions for people with chronic physical illness (White, 2000). More innovative approaches could also be explored, for example emotional disclosure (Kelley *et al.*, 1997; Smyth *et al.*, 1999) and Mindfulness meditation (Teasdale *et al.*, 2000).

The self-management programme used in the present study was relatively brief, when compared to those shown to be effective for the treatment of chronic pain (Morley, Eccleston & Williams, 1999). CBT interventions for people with early RA have also been much more intensive (Parker *et al.*, 1995). It is possible that more intensive programmes are needed to help people with chronic RA to make lasting changes in the self-regulation of their illness. Given the association between depression and poor health outcomes specific interventions could also be developed for the minority who experience clinically significant levels of depression. It would also be worth considering whether

psychological interventions for people with clinically significant levels of depression should be used in combination with anti-depressant medication.

There is some evidence to suggest that involving family members in psycho-educational interventions for people with rheumatic disease may be beneficial (Keefe *et al.*, 1996, 1999; Radojevic *et al.*, 1992; Taal, Rasker, Weigman, Brus, & Riemsma, 1994) but this issue needs further exploration.

The present study also suggests that there is a need to develop interventions that facilitate engagement in self-management and prevent relapse following treatment. The role of motivational factors has received little attention in the literature on psycho-educational intervention for people with rheumatic disease. For those who are less motivated to engage in self-management the use of methods developed to enhance motivation (e.g. motivational interviewing; Jensen, 2002; Miller & Rollnick, 2002) could be investigated further.

Effective methods for relapse prevention have been applied in a few intervention studies for people with RA (Parker *et al.*, 1995). Further work is needed to establish whether treatment packages incorporating comprehensive methods for preventing relapse are more effective in maintaining long-term gains.

10.7.5 How should interventions be delivered?

Most of the literature on psycho-educational interventions for people with rheumatic disease has investigated the use of specific structured group programmes led by health professionals or trained people with arthritis. Different methods for delivering interventions might provide an alternative for those people who do not wish to attend a group programme, or to prepare people for attending a group and to help them maintain gains after completing treatment.

Work is needed to increase health professionals understanding of the relevance of patients' illness representations for optimal engagement in medical care and self-management (Kaptein *et al.*, 2003). Incorporating the person's story about their illness into the clinical encounter is challenging. However, a better understanding of the interactions between how the person is cognitively and emotionally making sense of their illness experience, their efforts to cope and appraisals of coping efficacy could assist health professionals in achieving this. Training for health professionals led by "expert patients" has been shown to enhance retention of information, confidence and examination skills (Branch & Lipsky, 1998) and this approach could be developed to address psycho-social issues in rheumatic disease. As most people with RA have regular on-going contact with health services, the impact of training health professionals how to use approaches for enhancing motivation, self-regulation and relapse prevention in their routine consultations should be evaluated.

A different approach is suggested by a study showing that a mail delivered Arthritis Self-Management Programme along with individualised, computer-generated advice produced similar improvements to the group programme (Fries *et al.*, 1997). The use of computer-based psychological interventions for anxiety and depression has been receiving increased attention in recent years and this could be extended to work with people with chronic physical illness. Given the potential benefits, in terms of increased access and reduced costs, such interventions should be evaluated further.

10.7.6 Do interventions have other benefits or adverse effects?

Studies of psycho-educational intervention for rheumatic disease have failed to examine the impact on several important outcomes. These include reduction in drug toxicity, surgical intervention, work status and mortality. It is possible that currently available psycho-educational interventions will have little effect on these outcomes. However, a greater focus on them may encourage the development of novel interventions specifically targeting these outcomes. For example, Barlow *et al.*, (2001) have reported positive results from a preliminary evaluation of an intervention designed to reduced barriers to employment for

people with arthritis the "Into Work Personal Development (IWPD) programme. Although adverse reactions to psycho-educational interventions have not been reported no studies have explicitly attempted to identify these.

10.7.7 Leventhal's SRM

In relation to the SRM there are a number of areas for future research. Further longitudinal studies, involving experimental manipulation of self-regulatory processes, are needed to provide formal tests of the SRM assumptions about the inter-relationships between illness representations, coping and health outcomes (Hagger & Orbell, 2003; Kaptein *et al.*, 2003).

Future studies of the SRM might also benefit from focussing more on the extent to which self-regulatory processes predict behaviours and other objective measures (e.g. work function, use of health-care services, medication adherence, side-effects, medical complications or survival) rather than self-report measures (Kaptein *et al.*, 2003). For example, in RA a better understanding of the self-regulatory processes influencing medication use could have important implications for long-term health outcomes. In relation to this issue, future research might profitably explore representations of treatment. This is an area of current active research in other chronic illnesses (Horne, 2003). For example, in asthma beliefs about medication, rather than beliefs about asthma, were found to be important predictors of medication use (Horne & Weinman, 2002).

As discussed earlier, the empirical literature has also neglected emotional and appraisal processes and these need further attention (Cameron & Leventhal, 2003). Most of the studies using the SRM have investigated chronic illnesses. Studies on a broader range of illnesses, including acute illnesses, may facilitate the investigation of the moderating effects of illness type, severity and chronicity, on hypotheses generated from the SRM (Hagger & Orbell, 2003). Finally, the present study and much of the research using the SRM has tended to focus on the individual with the chronic illness. Further studies

investigating the influence of socio-cultural factors on the person's adaptation to RA, and other illnesses are needed (Weinman *et al.*, 2003)

Future research should also make use of different research designs and methods. In particular case studies may provide a low-cost way to evaluate novel interventions and to identify the essential components of treatment packages. Greater use of qualitative methods, such as focus groups and post-treatment interviews may also provide valuable insights into the processes of change occurring during treatment. Given the subtle dynamic interactions occurring during the process of self-regulation increased use of daily diary studies might also be helpful.

10.8 Summary of findings and conclusions

The study provides some evidence that a self-management intervention for hospital outpatients with chronic RA can improve health outcomes. Following the self-management intervention, the treatment group showed significantly greater improvements in individualized quality of life than the control group. The improvements were maintained at 9-month follow-up. The treatment group also showed short-term improvements in pain and depression immediately following the intervention, but these were not maintained at follow-up. The self-management intervention may not fully account for the improvements as some of the change occurred during the baseline phase.

There was stronger evidence that the intervention produced adaptive changes in illness-related cognitions and coping behaviours. Following the self-management intervention, the treatment group showed significantly greater improvements in self-efficacy for pain and other arthritis symptoms and practice of strengthening exercises, which were maintained at the 9-month follow-up. The treatment group also showed significantly greater improvements in illness identity, perceived consequences and use of planning as a coping strategy but these were not maintained at the 9-month follow-up.

Previous studies of self-management interventions for people with arthritis have found greater improvements in health outcomes. However, the findings are more consistent with studies of CBT for people with chronic RA. The results of this study indicate that the most likely explanation for the differences between the findings of self-management and CBT studies is not the type of intervention, but differences in the populations studied and the designs of the studies.

In the pooled sample, baseline illness representations were associated with coping procedures in the predicted directions. Perceiving RA to have multiple incoherent symptoms and serious consequences were associated with less use of adaptive and greater use of maladaptive coping strategies. Perceiving RA to be more controllable was associated with greater use of adaptive coping strategies.

In the cross-sectional analyses, at baseline, illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social function and disease activity).

In the longitudinal analyses, baseline illness representations and coping procedures predicted a significant amount of variance in future health outcomes (pain, disability, emotional distress, disease activity and health care utilization) at 3, 6 and/or 12 months.

Changes in illness representations and coping procedures explained a significant amount of variance in health outcomes (pain, disability, emotional distress, social function, occupational function, individualized quality of life and disease activity). Changes in illness representations and coping procedures were stronger and more consistent predictors of health outcomes than baseline illness representations and coping procedures.

Illness representations mediated the improvements in individualized quality of life and self-efficacy for pain and other arthritis symptoms found immediately following self-management intervention. Self-efficacy for pain and pain coping strategies moderated the maintenance of

treatment gains in individualized quality of life and self-efficacy for other arthritis symptoms respectively.

Further work is needed to develop and evaluate interventions for people with chronic RA that can demonstrate long-term improvements in health outcomes. The findings suggest, that focussing on approaches to facilitate the development of more adaptive illness and treatment representations and coping strategies might increase the effectiveness of interventions. In addition, greater attention to motivation, emotional regulation, appraisal and relapse prevention is needed.

The findings emphasise the importance of psychological factors in adaptation to chronic RA. The results support some of the assumptions underlying the SRM and are consistent with previous empirical research on chronic illness guided by the SRM. Further work is needed to explore neglected aspects of the self-regulatory process and to derive and test theoretical predictions from the model.

References

- Abdel- Nasser, A. M., Abd El-Azim, S., Taal, E., El-Badawy, S. A., Rasker, J. J., & Valkenburg, H. A. (1998). Depression and depressive symptoms in rheumatoid arthritis patients: An analysis of their occurrence and determinants. *British Journal of Rheumatology*, 37, 391-397.
- Abramson, L. Y., Seligman M. E. P., & Teesdale, J. P. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, 87, 49-74.
- Achterburg, J., McGraw, P., & Lawlis, G. F. (1981). Rheumatoid arthritis: A study of relaxation and temperature biofeedback training as an adjunctive therapy. *Biofeedback and Self Regulation*, 6, 207-223.
- Affleck, G., Pfeiffer, C., Tennen, H., & Fifield, J. (1987a). Attributional processes in rheumatoid arthritis patients. *Arthritis & Rheumatism*, 30, 927-931.
- Affleck, G., Pfeiffer, C., Tennen, H., & Fifield, J. (1988a). Social Support and Psychosocial Adjustment to Rheumatoid Arthritis: Quantitative and Qualitative Findings. *Arthritis Care and Research*, 1, 71-77.
- Affleck, G., Tennen, H., Pfeiffer, C. & Fifeld, J. (1987b). Appraisals of control and predictability in adapting to a chronic disease. *Journal of Personality and Social Psychology*, 53, 273-279.
- Affleck, G., Tennen, H., Pfeiffer, C. & Fifield, J. (1988b). Social Comparisons in Rheumatoid Arthritis: Accuracy and adaptational significance. *Journal of Social and Clinical Psychology*, 6, 219-234.
- Affleck, G., Tennen, H., Pfeiffer, C., Fifield, J. & Rowe, J. (1987c). Downward comparison and coping with serious medical problems. *American Journal of Orthopsychiatry*, 57, 570-577.
- Affleck, G., Tennen, J., Urrows, S. & Higgins, P. (1992a). Neuroticism and the pain-mood relation in RA: insights from a prospective daily study. *Journal of Consulting & Clinical Psychology*, 60, 119-126.
- Affleck, G., Urrows, S., Tennen, H. & Higgins, P. (1992b). Daily coping with pain from rheumatoid arthritis: patterns and correlates. *Pain*, 51, 221-229.
- Affleck, G., Urrows, S., Tennen, H., Higgins, P., Pav, D. & Aloisi, R. (1997). A dual pathway model of daily stressor effects on rheumatoid arthritis. *Annals of Behavioural Medicine*, 19, 161-170.
- Albano, S. A., Santana-Sahagun, E. & Weisman, M. H. (2001). Cigarette smoking and rheumatoid arthritis. *Seminars in Arthritis & Rheumatism*. 31, 146-159.

- Allaire, S. H. (1996). Work Disability. In S. T. Wegener, B. L. Belza, & E. P. Gall (Eds.), *Clinical Care in the Rheumatic Diseases* (pp. 141-145). Atlanta: The American College of Rheumatology.
- Allaire, S. H., Meenan, R. F. & Anderson, J. J. (1991). The impact of RA on the household work performance of women. *Arthritis & Rheumatism*, 34, 669-678.
- Allaire, S. H., Prashker, M. & Meenan, R. F. (1994). The costs of rheumatoid arthritis. *Pharmoeconomics*, 6, 513-522
- Alloy, L. B, Abramson, L. Y, Metalsky, G. I. & Hartlage, S (1988). The hopelessness theory of depression: Attributional aspects. *British Journal of Clinical Psychology*, 27(1), 5-21.
- Anderson, J. J., Firschein, H. E. & Meenan, R. F. (1989). Sensitivity of a health status measure to short-term clinical changes in arthritis. *Arthritis and Rheumatism*, 32, 844-850.
- Anderson, K. O., Bradley, L. A., Young, L. D., McDaniel, L. K. & Wise, C. M. (1985). Rheumatoid Arthritis: Review of Psychological Factors Related to Etiology, Effects and Treatment. *Psychological Bulletin*, 98, 358-387.
- Anderson, K. O., Keefe, F. J., Bradley, L. A., McDaniel, L. K., Young, L. D., Turner, R. A. et al., (1988). Prediction of pain behaviour and functional status of rheumatoid arthritis patients using medical status and psychological variables. *Pain*, 33, 25-32.
- Applebaum, K. A., Blanchard, E. B., Hickling, E. B., Hickling, E. J. & Alfonso, M. (1988). Cognitive Behavioural treatment of a veteran population with moderate to severe rheumatoid arthritis. *Behaviour Therapy*, 19, 489-502
- Arnett, F. C., Edworthy, S. M., Bloch, D. A., McShane, D. J., Fries, J. F., Cooper, N. S., Healey, L. A., Kaplan, S. R., Liang, M. H., Luthra, H. S., et al., (1988). The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis & Rheumatism*, 31, 315-24.
- Arthritis Foundation (2003). Rheumatoid Arthritis. Retrieved June 9th, 2003 from <http://www.arthritis.org/conditions/diseasecenter/ra.asp#Who%20Is%20At%20Risk>
- Arthritis Research Campaign (2002). Arthritis The Big Picture. Retrieved October 1st, 2002, from <http://www.arc.org.uk>
- Aron, A. & Aron, E. N. (1999). *Statistics For Psychology (2nd Edn)*. London: Prentice-Hall.
- Astin, J. A., Beckner, W., Soeken, K., Hochberg, M. C. & Berman, B. (2002). Psychological interventions for rheumatoid arthritis: A meta-analysis of randomised controlled trials. *Arthritis Care & Research*, 47, 291-302.

- Badley, E. M. & Tenant, A. (1993). Disablement Associated with Rheumatic Disorders in a British Population: Problems with Activities of Daily Living and Level of Support. *British Journal of Rheumatology*, 32, 601-608.
- Baker, F. (1953). The rationale for physical therapy in arthritis. *Bull. Rheum. Dis.*, 4, 57-58.
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioural change. *Psychological Review*, 84, 191-215.
- Banks, R. E., Whicher, J. T., Thompson, D., & Bird, H. A. (1998). Acute Phase Response. In P. J. Maddison, D. A. Isenberg, P. Woo & D. N. Glass (Eds.). *Oxford Textbook of Rheumatology*, (2nd Ed Vol 1 pp. 63-72). New York: Oxford University Press.
- Barlow, J. H. (2001a). Arthritis. In D. W. Johnston & M. Johnston (Eds.), *Health Psychology, Comprehensive Clinical Psychology*: (Vol. 8., pp. 427-443), Elsevier Science Publishers B.V.
- Barlow, J. H. (2001b). How to use education as an intervention in osteoarthritis. In M. Doherty & M. Dougados (Eds.), Osteoarthritis. Balliere's best practice. *Research Clinical Rheumatology*, 15, 545-558.
- Barlow, J. H., Cullen, L., Davis, S., & Williams, B. (1997a). The power of sharing common experiences: The "hidden" benefit of group education for people with arthritis. *British Journal of Therapy and Rehabilitation*, 4, 38-41.
- Barlow, J.H., Cullen, L. A., & Rowe, I. F. (1999a). Comparison of knowledge and psychological well-being between patients with a short disease duration (< or = 1 year) and patients with more established rheumatoid arthritis (> or = 10 years duration). *Patient Education and Counselling*, 1999, 38, 195-203.
- Barlow, J. H., Cullen, L. A., & Rowe, I. F. (2002a). Educational preferences, psychological well-being and self-efficacy among people with rheumatoid arthritis. *Patient Education & Counseling*. 46, 11-9.
- Barlow, J. & Harrison, K. (1996). Focusing on empowerment: Facilitating self-help in young people with arthritis through a disability organisation. *Disability & Society*, 11, 539-551.
- Barlow, J. H., Shaw, K. L., & Harrison, K. (1999c). Consulting the 'experts': Children's and parents' perceptions of psycho-educational interventions in the context of juvenile chronic arthritis. *Health Education Research*, 14, 597-610.
- Barlow, J. H., Turner, A. P., & Wright, C. C. (1998b). A longer term follow-up of an arthritis self-management programme. *British Journal of Rheumatology*, 37, 1315-1319.
- Barlow, J. H., Turner, A. P., & Wright, C. C. (1998a). Sharing, caring and learning to take control: self-management training for people with arthritis. *Psychology, Health & Medicine*, 3, 387-393.

- Barlow, J. H., Turner, A. P., & Wright, C. C. (2000). A randomized controlled study of the Arthritis Self-Management Programme in the UK. *Health Education Research*, 15, 665-680.
- Barlow, J. H., Williams, R. B., & Wright, C. (1997c). The Arthritis Self-Efficacy Scale in a UK context Psychology. *Health & Medicine*, 2, 5-19.
- Barlow, J. H., Williams, B., & Wright, C. (1996). The Generalized Self-Efficacy Scale in people with arthritis. *Arthritis Care & Research*, 9, 189-96.
- Barlow, J. H., Williams, B., & Wright, C. (1997b). Improving arthritis self-management in older adults: 'Just what the doctor didn't order'. *British Journal of Health Psychology*, 2, 175-185.
- Barlow, J. H., Williams, B., & Wright, C. (1999b). Instilling the strength to fight the pain and get on with life: learning to become an arthritis self-manager through an adult education programme. *Health Education & Research*, 14, 533-544.
- Barlow, J., Wright, C., & Kroll, T. (2001). Overcoming perceived barriers to employment among people with arthritis. *Journal of Health Psychology*, 6, 205-216.
- Barlow, J., Wright, C., Sheasby, J., Turner, A., & Hainsworth, J. (2002b). Self-management approaches for people with chronic conditions: a review. *Patient Education and Counselling*, 48, 177-187.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, 51, 1173-1182.
- Beckham, J. C., D'Amico, C. S., Rice, J. R., Jordan, J. S., Divine, G. W., & Brook, W. B. (1992). Depression and level of functioning in patients with rheumatoid arthritis. *Canadian Journal of Psychiatry*, 37, 539- 543.
- Beery, H., & Huskinson, E. (1972). Measurement of pain. *Journal of Clinical Trials*, 9, 13-18.
- Belza, B. L. (1996). Fatigue. In S. T. Wegener, B. L. Belza, & E. P. Gall (Eds.), *Clinical Care in the Rheumatic Diseases* (pp. 117-120). Atlanta: The American College of Rheumatology.
- Berkanovic, E., Oster, P., Wong, W. K., Bulpitt, K. et al., (1996). The relationship between socioeconomic status and recently diagnosed rheumatoid arthritis. *Arthritis Care & Research*, 9, 457-462.
- Bishop, G. D. (1991). Understanding the understanding of illness. Lay disease representations. In J. A. Skelton & R. T. Croyle (Eds.), *Mental Representations in Health and Illness* (pp. 32-59). New York Inc: Springer-Verlag.
- Blalock, S.J., DeVellis, B.M., Holt, K., & Hahn, P.M. (1993). Coping with rheumatoid arthritis: is one problem the same as another? *Health Education Quarterly*, 20, 119-132.

- Borg, G, Allander, E. Berg, E. Brodin, U., From, A., & Trang, L. (1988). Auranofin improves outcome in early rheumatoid arthritis. Results from a 2 year, double blind, placebo controlled study. *Journal of Rheumatology*, 15, 1747-1754.
- Boutaugh, M. L., & Lorig, K. R. (1996). Patient Education. In S. T. Wegener, B. L. Belza, & E. P. Gall (Eds.), *Clinical Care in the Rheumatic Diseases* (pp. 53-58). Atlanta: The American College of Rheumatology.
- Bradley, L. A. (1989). Adherence with treatment regimens among adult rheumatoid arthritis patients: Current status and future directions. *Arthritis Care & Research*, 2, 33-39.
- Bradley, L. A., & Alberts, K. R. (1999). Psychological and behavioural approaches to pain management for patients with rheumatic disease. *Rheumatic Disease Clinics of North America*, 25, 215-232.
- Bradley, L. A., Young, L. D., Anderson, K. O., Turner, R. A., Agudelo, C. A., McDaniel, L. K., Pisko, E. J., Semble, E. L., & Morgan, T. M. (1987). Effects of psychological therapy on pain behavior of rheumatoid arthritis patients: treatment outcome and six-month followup. *Arthritis Rheum*, 30, 1105-14.
- Branch, V. K., & Lipsky, P. E. (1998). Positive impact of an intervention by arthritis educators on retention of information, confidence, and examination skills of medical students. *Arthritis Care & Research*, 11, 32-38.
- Brekke, M., Hjortdahl, P., Thelle, D. S., & Kvien, T. K. (1999). Disease activity and severity in patients with rheumatoid arthritis: Relations to socioeconomic inequality. *Social Science & Medicine*, 48, 1743- 1750.
- Bresnihan, B. (2002) Rheumatoid Arthritis: Principles of Early Treatment. *Journal of Rheumatology*, 29, 9-12.
- Brooks, P., & Hochberg, M. (2001). Outcome measures and classification criteria for the rheumatic diseases. A compilation of data from OMERACT (Outcome Measures for Arthritis Clinical Trials), ILAR (International League of Associations for Rheumatology), regional leagues and other groups. *Rheumatology*, 40, 896-906.
- Brown, G. K. (1990). A causal analysis of chronic pain and depression. *Journal of Abnormal Psychology*, 99, 127-137.
- Brown, G. K., & Nicassio, P. M. (1987). Development of a questionnaire for the assessment of active and passive coping strategies in chronic pain patients. *Pain*, 31, 53-64.
- Brown, G., Nicassio, P., & Wallston, K. (1989a). Pain coping strategies and depression in Rheumatoid Arthritis. *Journal of Consulting & Clinical Psychology*, 57, 652-657.
- Brown, G. K., Wallston, K. A., & Nicassio, P. M. (1989b) Social Support and depression in rheumatoid arthritis: A one-year prospective study. *Journal of Applied Social Psychology*, 19, 1164-1181.

- Brown, M. A., & Wordsworth, B. P. (1998). Genetic studies of common rheumatological diseases. *British Journal of Rheumatology*, 37, 818-23.
- Brus, H. L. M., Taal, E., van de Laar, M. A. F. J., Rasker, J. J. & Wiegman, O. (1997). Patient education and disease activity: A study among rheumatoid arthritis patients. *Arthritis Care & Research*, 10, 320-324.
- Brus, H.L.M., van de Laar, M., Taal, E., Rasker, J., & Wiegman, O. (1999). Determinants of compliance with medication in patients with rheumatoid arthritis: The importance of self-efficacy expectations. *Patient Education & Counseling*, 36, 57-64.
- Buckley, C.D. (1997). Science, medicine and the future: Treatment of rheumatoid arthritis. *British Medical Journal*, 315, 236-238.
- Buescher, K. L., Johnston, J. A., Parker, J. C., Smarr, K. L., Buckelew, S. P., Anderson, S. K., & Walker, S. E. (1991). Relationship of self-efficacy to pain behaviour. *Journal of Rheumatology*, 18, 968-972.
- Buick, D. L. (1997). Illness representations and breast cancer: Coping with radiation and chemotherapy. In K.J. Petrie & J. Weinman (Eds.), *Perceptions of health and illness: Current research and applications* (pp. 379-409). Amsterdam: Harwood Academic Publishers.
- Buick, D. L., & Petrie, K. J. (2002). "I know just how you feel": The validity of healthy women's perceptions of breast cancer patients' distress, coping and illness perceptions during radiation and chemotherapy treatment. *Journal of Applied Social Psychology*, 32(1), 110-123.
- Burckhardt, C. S. (1994). Arthritis and musculoskeletal patient education standards. *Arthritis Care & Research*, 7, 1-4.
- Burckhardt, C. S., Archenholtz, B., & Bjelle, A. (1993). Quality of life of women with systemic lupus erythematosus: a comparison with women with rheumatoid arthritis. *Journal of Rheumatology*, 20, 977-81.
- Callahan, L. F., Bloch, D. A., & Pincus, T. (1992). Identification of work disability in rheumatoid arthritis: Physical, radiographic and laboratory variables do not add explanatory power to demographic and functional variables. *Journal of Clinical Epidemiology*, 45, 127-138.
- Callahan, L. F., Cordray, D. S., Wells, G., & Pincus, T. (1996). Formal education and five-year mortality in rheumatoid arthritis: Mediation by helplessness scale scores. *Arthritis Care & Research*, 9, 463-472.
- Callahan, L. F., Pincus, T., Huston, J. W., Brooks, R. H., Nance, E. P. Jr., & Kaye, J. J. (1997). Measures of activity and damage in rheumatoid arthritis: depiction of changes and prediction of mortality over five years. *Arthritis Care & Research*, 10, 381-94.

Cameron, L. D., Leventhal, H., & Love, R. R. (1998). Trait anxiety, symptom perceptions, and illness-related responses among women with breast cancer in remission during a tamoxifen clinical trial. *Health Psychology, 17*, 459-469.

Cameron, L. D., & Leventhal, H. (2003). Self-regulation of health and illness: an overview. In L. D. Cameron & H. Leventhal.(Eds.) *The self-regulation of health and illness behaviours*. New York: Routledge.

Carr, A., & Thompson, P. W. (1994). Towards a measure of patient-perceived handicap in RA. *British Journal of Rheumatology, 33*, 378-382.

Carver, C. S. (1999). You want to measure coping but your protocol's too long: Consider the Brief COPE. *International Journal of Behavioral Medicine, 4* (1), 92-100.

Carver, C. S., Pozo, C., Harris, S. D., Noriega, V., Scheier, M. F., Robinson, D. S., Ketcham, A. F., Moffat, F. L. Jr., & Clark, K. C. (1993). How coping mediates the effect of optimism on distress: A study of women with early stage breast cancer. *Journal of Personality & Social Psychology, 65*, 375-390.

Carver, C. S., & Scheier, M. F. (1996). *Perspectives on personality*, (3rd ed.). Boston MA: Allyn and Bacon.

Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing Coping Strategies: A Theoretical Based Approach. *Journal of Personality and Social Psychology, 56*, 267-283.

Clarke, A., & Fallowfield, L. J. (1986). Quality of life measurement in patients with malignant disease: a review. *Journal of the Royal Society of Medicine, 79*, 165-168.

Clarke, A. E. (1997). Arthritis patient education: How economic evaluations can inform health policy. *Canadian Journal on Aging, 16*, 162-176.

Cohen, J. L., Sauter, S. V., DeVellis, R. F., & DeVellis, B. M. (1986). Evaluation of arthritis self-management courses led by lay persons and by professionals. *Arthritis Rheum. 29*, 388-393.

Cooper, A., Lloyd, G. S., Weinman, J. & Jackson, G. (1999). Why patients do not attend cardiac rehabilitation: Role of intentions and illness beliefs. *Heart, 82*, 234-236.

Compass, B. E., Haaga, D. A., Keefe, F. J., Leitenberg, H., & Williams, D. A. (1998). Sampling of empirically supported psychological treatments for health psychology: Smoking, chronic pain, cancer, and bulimia nervosa. *Journal of Consulting and Clinical Psychology, 66*, 89-112.

Contrada, R. J. & Ashmore, R. D. (Eds.) (1999). *Self and social identity vol 2: self, social identity and physical health*, New York: Oxford University Press.

- Cornelissen, P. G. J., Rasker, J. J., & Valkenburg, H. A. (1988). The arthritis sufferer and the community: a comparison of arthritis sufferers in rural and urban areas. *Ann. Rheum. Dis.*, 47, 150-156.
- Cosh, J. A. (1984). Survival and death in rheumatoid arthritis. *Journal of Rheumatology*, 1, 17-19.
- Coyne, J. C., & Racioppo, M. W. (2000). Never the twain shall meet? Closing the gap between coping research and clinical intervention research. *American Psychologist*, 55, 655-664.
- Crawford, J. R., Henry, J. D., Crombie, C., & Taylor, E. P. (2001). Normative data for the HADS from a large non-clinical sample. *British Journal of Clinical Psychology*, 40, 429-434.
- Creed, F., & Ash, G. (1992). Depression in rheumatoid arthritis: Aetiology and treatment. *International Review of Psychiatry*, 4, 23-33.
- Crisson, J. E., & Keefe, F. J. (1988). The relationship of locus of control to pain coping strategies and psychological distress in chronic pain patients'. *Pain*, 35, 147-54.
- Crofford, L. J., & Casey, K. L. (1999). Central modulation of pain perception. *Rheumatic Disease Clinics of North America*, 25, 1-13.
- Crosby, L. F. (1991). Factors which contribute to fatigue associated with rheumatoid arthritis. *Journal of Advanced Nursing*, 16, 974-981.
- Daltroy, L. H. (1993). Doctor-patient communication in rheumatological disorders. *Baillieres Clinical Rheumatology*, 7, 221-239.
- Daltroy L. H., & Liang M. H. (1993). Arthritis education: Opportunities and state of the art. *Health Education Quarterly*, 20, 3-16.
- Davis, P., Busch, A. J., Lowe, J. C., Taniguchi, J., & Dikowich, B. (1994). Evaluation of a rheumatoid arthritis patient education program: impact on knowledge and self-efficacy. *Patient Education and Counselling*, 24, 55-61.
- Dempster, M., Donnelly, M., & Fitzsimons, D. (2002) Generic, disease-specific and individualised approaches to measuring health-related quality of life among people with heart disease – A comparative analysis. *Psychology and Health*, 17, 447-457.
- Department for Work and Pensions. (2001). *Analytical Services Division*. Retrieved April 30, 2002, from the www.dwp.gov.uk
- Department of Health. (1999). *Saving lives: our healthier nation*. London: HMSO.
- Department of Health. (2001). *The Expert Patient: A New Approach to Chronic Disease Management for the 21st Century*. London: Department of Health Publications. Retrieved December 1st, 2002, from www.ohn.gov.uk/ohn/people/expert.htm.

Department of Health. (n.d.). Hospital Episode statistics 1999/2000 (England). Retrieved February 24, 2003,, from <http://www.doh.gov.uk>

Dessein, P. H., Shipton, E. A., & Budd, K. (2000). Nociceptive and non-nociceptive rheumatological pain: Recent developments in the understanding of pathophysiology and management in rheumatoid arthritis and fibromyalgia, *Pain Review*, 7, 67-79.

DeVellis, B. M. (1993). Depression in rheumatological diseases. *Balliere's Clinical Rheumatology*, 7, 241-258.

DeVellis, B. M., Blalock, S. J., Hahn, P. M., Devellis, R. F., & Hochbaum, G. M. (1988). Evaluation of a Problem-Solving Intervention for Patients With Arthritis. *Patient Education and Counselling*, 11, 29-42.

DeVellis, R., & Blalock, S. (1993). Psychological and educational interventions to reduce arthritis disability. *Bailleres Clinical Rheumatology*, 7, 397-416.

DeVellis, R. F., & Callahan, L. F. (1993). A brief measure of helplessness in rheumatic disease: the helplessness subscale of the Rheumatology Attitudes Index. *Journal of Rheumatology*, 20, 866-869.

DeVellis, R. F., Holt, K., Renner, B. R., Blalock, S. J., Blanchard, L.W. *et al.*, (1990). The relationship of social comparisons to rheumatoid arthritis symptoms and affect. *Basic and Applied Social Psychology*, 11, 1-18.

Devins, G. M., Edworthy, S. M., Guthrie, N. G., & Martin, L. (1992). Illness intrusiveness in rheumatoid arthritis: Differential impact on depressive symptoms over the adult lifespan. *Journal of Rheumatology*, 19, 709- 715.

Devlin, J., Gough, A., Huissoon, A., Perkins, P., Holder, R., Reece, R., Arthur, V. & Emery, P. (1997). The acute phase and function in early rheumatoid arthritis. C-reactive protein levels correlate with functional outcome. *Journal of Rheumatology*, 24, 9-13.

Deyo, R.A. (1988) Measuring quality of life of patients with rheumatoid arthritis. In S. R. Walker and R. M. Rosser (Eds.), *Quality of life: assessment and application* (pp. 205). MTP, Lancaster.

Diefenbach, M. A., & Leventhal, H. (1996). The common-sense model of illness representation: Theoretical and practical considerations. *J. Soc. Distress Homeless*, 5, 11-38.

Dixon, J. S. Hayes, S., Constable, P. D., & Bird, H. A. (1988). What are the 'best' measurements for monitoring patients during short-term second-line therapy? *British Journal of Rheumatology*, 27, 37-43.

Doherty, M., Brandt, K., & Lohmander, S. (1998). *Textbook on osteoarthritis*. London: Oxford University Press.

Donovan, J. (1991). Patient education and the consultation: the importance of lay beliefs. *Annals of the Rheumatic Diseases*, 50, 418-421.

Doeglas, D., Suurmeijer, T., Krol, B., Sanderma, R., van Leeuwen, M. & van Rijswijk M. (1995). Work disability in early rheumatoid arthritis. *Annals of the rheumatic diseases*, 54, 455-60.

Doeglas, D., Suurmeijer, T., Krol, B., Sanderma, R., van Rijswijk, M., & van Leeuwen, M. (1994). Social support, social disability, and psychological well-being in rheumatoid arthritis. *Arthritis Care & Research*, 7, 10-15.

Downe-Wamboldt, B. L., & Melanson, P. M. (1995). A causal model of coping and well-being in elderly people with arthritis. *Journal of Advanced Nursing*, 27, 1109-1116.

Drossaers-Bakker, K. W., de Buck, M., van Zeben, D., Zwinderman, A. H., Breedveld, F. C., & Hazes, J. M. W. (1999). Long-term course and outcome of functional capacity in rheumatoid arthritis: the effect of disease activity and radiologic damage over time. *Arthritis and Rheumatism*, 42, 1854-60.

Eberhardt, K., & Fex, E. (1998). Clinical course and remission rate in patients with early rheumatoid arthritis: relationship to outcome after 5 years. *British Journal of Rheumatology*, 37, 1324-9.

Eberhardt, K., Fex, E., Johnson, U., & Wolheim, F. A. (1996). Associations of HLA-DRB and -DQB genes with two and five year outcome in rheumatoid arthritis. *Annals of Rheumatic Diseases*, 55, 34-9.

Eberhardt, K., Larson, B., & Nived, K. (1993). Early RA - some social, economic and psychological aspects. *Scandinavian Journal of Rheumatology*, 22, 119-124.

Eberhardt, K. B., Truedsson, L., Pettersson, H., Svensson, B., Stigsson, L., Eberhardt, J. L., & Wollheim, F. A. (1990). Disease activity and joint damage progression in early rheumatoid arthritis: relation to IgA, IgA and IgM rheumatoid. *Annals of the Rheumatic Disease*, 49, 906-909.

Edwards, J. C. W. (2002) Pathogenesis of Rheumatoid Arthritis. Arthritis Research Campaign: Topical Reviews, Recovered, May 8, 2002, from <http://www.arc.org.uk>

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196, 129-136.

Erhardt, C. C., Mumford, P. A., Venables, P.J., & Maini, R. N. (1989). Factors predicting a poor life prognosis in rheumatoid arthritis: an eight year prospective study. *Annals of Rheumatic Diseases*, 48, 7-13.

Escalante, A., & Del Rincon, I. (1999). How much disability in rheumatoid arthritis is explained by rheumatoid arthritis? *Arthritis & Rheumatism*, 42, 1712-1721.

Evers, A.W.M., Kraaijmaat, F.W., Geenen, R., & Bijlsma, J. W. (1997). Determinants of psychological distress and its course in the first year after diagnosis in rheumatic arthritis patients. *Journal of Behavioral Medicine*, 20, 489-504.

- Evers, A. W. M., Kraaimaat, F. W., Geenen, R., & Bijlsma, W. J. (1998). Psychosocial predictors of functional change in recently diagnosed rheumatoid arthritis patients. *Behaviour, Research and Therapy*, 36, 179-193.
- Felson, D. T., Anderson, J. J., Boers, M., Bombardier, C., Chernoff, M., Fried, B., Furst, D., Goldsmith, C., Kieszak, S., Lightfoot, R., *et al.*, (1993). The American College of Rheumatology preliminary core set of disease activity measures for rheumatoid arthritis clinical trials. The Committee on Outcome Measures in Rheumatoid Arthritis Clinical Trials. *Arthritis & Rheumatism*, 36, 729-40.
- Felton, B. J., & Revenson, T. A. (1984). Coping with chronic illness: a study of illness controllability and the influence of coping strategies on psychological adjustment. *Journal of Consulting and Clinical Psychology*, 52, 343-353.
- Fernandez, E., & Milburn, T. W. (1994). Sensory and affective predictors of overall pain and emotions associated with affective pain. *Clinical Journal of Pain*, 10, 3-9.
- Fex, E., Jonsson, K., Johnson, U., & Eberhardt, K. (1996). Development of radiographic damage during the first 5-6 yr of rheumatoid arthritis. A prospective follow-up study of a Swedish cohort. *British Journal of Rheumatology*, 35, 1106-1115.
- Fife-Schaw, C. (2000) . Levels of measurement. In: Breakwell, G. M. Hammond, S. and Fife- Schaw, C. (Eds.). *Research Methods in Psychology*, (2nd Ed). Sage, London.
- Fifield, J., Reisine, S., & Grady, K. (1991). Work disability and the experience of pain and depression in RA. *Social Science & Medicine*, 33, 579-585.
- Fifield, J., Tennen, H., Reisine, S., & McQuillan, J. (1998). Depression and the long term risk of pain, fatigue, and disability in patients with rheumatoid arthritis. *Arthritis and Rheumatism*, 41, 1851-1857.
- Firestein, G. S. (1996). Invasive fibroblast-like synoviocytes in rheumatoid arthritis: passive responders or transformed aggressors? *Arthritis & Rheumatism*, 39, 1781-90.
- Fitzgerald, O., & Bresnihan, B. (1983). Patients with rheumatoid arthritis first presenting to a rheumatology unit with severe established disability. *Ir. J. Med. Sci.*, 152, 268-71.
- Fitzpatrick, R., Newman, S., Archer, R. & Shipley, M. (1991) Social support, disability and depression: A longitudinal study. *Social Science & Medicine*, 33, 605-611.
- Fitzpatrick, R., Newman, S., Jenkinson, C., & Mowat, A. (1992). A generic health status instrument in the assessment of rheumatoid arthritis. *British Journal of Rheumatology*, 31, 87-90.
- Fitzpatrick, R., Newman, S., Lamb, R. & Shipley, M. (1988). Social relationships and psychological well-being in rheumatoid arthritis. *Social Science Medicine*, 27, 399-403.

Flor, H., Haag, G., Turk, D. C., & Koehler, H. (1983). Efficacy of EMG biofeedback, pseudotherapy, and conventional medical treatment for chronic rheumatic back pain. *Pain, 17*, 21-31.

Flor, H., & Turk, D. C. (1988). Chronic back pain and rheumatoid arthritis predicting pain and disability from cognitive variables. *Journal of Behavioural Medicine, 11*, 251-265

Folkman, S., & Lazarus, R. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behaviour, 21*, 219-239.

Folkman, S., & Lazarus, R. (1986). Stress processes and depressive symptomatology. *Journal of Abnormal Psychology, 95*, 107-113.

Fortune, D. G., Richards, H. L., Main, C. J., & Griffiths, C. E. M. (2000). Pathological worrying, illness perceptions and disease severity in patients with psoriasis. *British Journal Of Health Psychology, 5*, 71-82.

Frank, R. G., & Hagglund, K. J. (1996). Mood Disorders. In S. T. Wegener, B. L. Belza, & E. P. Gall (Eds.), *Clinical Care in the Rheumatic Diseases* (pp. 125-130). Atlanta: The American College of Rheumatology.

Fries, J. F. (1995). *Arthritis: a take care of yourself health guide for understanding your arthritis*. (4th Ed.) Perseus Books: Reading, Massachusetts.

Fries, J. F., & Bellamy, N. (1991). Introduction. In N. Bellamy (Ed.), *Prognosis in the Rheumatic Diseases*, (pp.1-10). London: Kluwer.

Fries, J. F., Carey, C., & McShane, D. J. (1997). Patient education in arthritis: randomized controlled trial of a mail-delivered program. *Journal of Rheumatology, 24*, 1378-1383.

Fries, J. F., Spitz, P., Kraines, R. G., & Holman, H. R. (1980). Measurement of patient outcome in Arthritis. *Arthritis & Rheumatism, 23*, 137-45.

Fries J. F., Williams, C. A., Morfeld, D., Singh, G., & Sibley, J. (1996). Reduction in long-term disability in patients with rheumatoid arthritis by disease-modifying antirheumatic drug-based treatment strategies. *Arthritis & Rheumatism, 39*, 616-622.

General Register Office for Scotland. (2000). Causes of Death: 2000 Annual report. Recovered 12 January, 2003, from www.gro-scotland.gov.uk

Germond, S., Schomer, H. H., Meyers, O. L., & Weight, L. (1993). Pain management in rheumatoid arthritis: a cognitive-behavioural intervention. *South African Journal of Psychology, 23*, 1-9.

Gibson, T., & Clark, B. (1985) Use of simple analgesics in rheumatoid arthritis. *Annals of the Rheumatic Diseases, 44*, 27-29.

- Goeppinger, J., Arthur, M. W., Baglioni, A. J. Jr., Brunk, S. E., & Brunner, C. M. (1989). A reexamination of the effectiveness of self-care education for persons with arthritis. *Arthritis & Rheumatism*, 32, 706-716..
- Gotzsche, P. C. (1989). Methodology and overt and hidden bias in reports of 196 double-blind trials of nonsteroidal antiinflammatory drugs in rheumatoid arthritis. *Control Clin Trials*, 10, 31-56.
- Gough, A., Faint, J., Salmon, M., Hassell, A., Wordsworth, P., Pilling, D., Birley, A., & Emery, P. (1994). Genetic typing of patients with inflammatory arthritis at presentation can be used to predict outcome. *Arthritis & Rheumatism*, 37, 1166-1170.
- Green, L. W., & Kreuter, M. W. (1991). *Health promotion planning, an educational and environmental approach*. Mountain View, California: Mayfield.
- Griva, K., Myers, L. B., & Newman, S. (2000). Illness perceptions and self-efficacy beliefs in adolescents and young adults with insulin dependent diabetes mellitus. *Psychology and Health*, 15, 733-750.
- Groessl, E. J., & Cronan, T. A. (2000). A cost analysis of self-management programs for people with chronic illness. *American Journal of Community Psychology*, 28, 455-480.
- Hagger, M. S., & Orbell, S. (2003). A meta-analytic review of the common-sense model of illness representations. *Psychology and Health*, 18 (2), 141-184
- Halldorsdottir, H. D., Jonsson, T., Thorsteinsson, J., & Valdimarsson, H. (2000). A prospective study on the incidence of rheumatoid arthritis among people with persistent increase of rheumatoid factor. *Annals of Rheumatic Diseases*, 59, 149-51.
- Hammond, A., Lincoln, N., & Sutcliffe, L. (1999). A crossover trial evaluating an educational-behavioural joint protection programme for people with rheumatoid arthritis. *Patient Education & Counseling*, 37, 19-32.
- Hampson, S. E., Glasgow, R. E., & Toobert, D. J. (1990). Personal models of diabetes and their relations to self-care activities. *Health Psychology*, 5, 632-646.
- Hampson, S. E., Glasgow, R. E., & Zeiss, A. (1994). Personal models of OA and their relation to self-management activities and quality of life. *Journal of Behavioural Medicine*, 17, 143-158.
- Harris, E. (1993). Clinical features of rheumatoid arthritis. In W.N. Kelley, E.D. Harris, S. Ruddy, & C. B. Sledge (Eds.), *Textbook of rheumatology* (pp. 122-154). Philadelphia: Saunders.
- Hawley, D. J. (1995). Psycho-educational interventions in the treatment of arthritis. *Baillere's Clinical Rheumatology*, 9, 803-823.
- Hawley, D. J., & Wolfe, F. (1988). Anxiety and depression in patients with rheumatoid arthritis: a prospective study of 400 patients. *Journal of Rheumatology*, 15, 932-941.

- Hawley, D. J., & Wolfe, F. (1993). Depression is not more common in rheumatoid arthritis: 10 Year longitudinal study of 6153 patients with rheumatic diseases. *Journal of Rheumatology*, 20, 2025-2031.
- Hazes, J. M. W., & Silman, A. J. (1990) Review of UK data on the rheumatic diseases-2. Rheumatoid arthritis. *British Journal of Rheumatology*, 29, 310-2.
- Haywood, K. L., Garratt, A. M., Dziedzic, K., & Dawes, P. T. (2003). Patient Centered assessment of ankylosing spondylitis-specific health related quality of life: evaluation of the patient generated index. *Journal of Rheumatology*, 30, 764-773.
- Heijmans, M. (1998). Coping and adaptive outcome in chronic fatigue syndrome: importance of illness cognitions. *Journal of Psychosomatic Research*, 45, 39-51.
- Heijmans, M. (1999). The role of patients' illness representations in coping and functioning with Addison's disease. *British Journal of Health Psychology*, 4, 137-149.
- Heijmans, M., & de Ridder, D. (1998). Assessing illness representations of chronic illness: Explorations of their disease- specific nature. *Journal of Behavioral Medicine*, 21, 485-503.
- Heijmans, M., de Ridder, D. & Bensing, J. (1999). Dissimilarity in patients' and spouses representations of chronic illness: exploration of relations to patient adaptation. *Psychology and Health*, 14, 451-466.
- Henderson, S. E., & McMillan, I. R. (2002). Pain and function: Occupational therapists' use of orthotics in rheumatoid arthritis. *British Journal of Occupational Therapy*, 65, 165-171.
- Herd, R. M., Tidman, M. J., Ruta, D. A., & Hunter, J. A. (1997). Measurement of quality of life in atopic dermatitis: correlation and validation of two different methods. *British Journal of Dermatology*, 136, 502-507.
- Herrmann, C. (1997). International experiences with the Hospital Anxiety and Depression Scale - A review of validation data and clinical results. *Journal of Psychosomatic Research*, 42, 17-41.
- Hicks, J. E. (1990). Exercise in patients with inflammatory arthritis and connective tissue disease. *Rheum. Dis. Clin. North Am.*, 16, 845-870.
- Hirano, P. C., Laurent, D. D., & Lorig, K. (1994). Arthritis patient education studies, 1987-1991: a review of the literature. *Patient Education and Counselling* 24, 9-54.
- Holman, V., & Lorig, K. (1992). Perceived self efficacy in self management of chronic disease. In Schwarzer R. (Ed.), *Self efficacy: Thought control of action*. Hemisphere, Washington DC.
- Horne, R. (2003). Treatment perceptions and self-regulation. In L.D. Cameron, & H. Leventhal (eds.). *The self-regulation of health and illness behaviours*. New York: Routledge.

Horne, R., & Weinman, J. (2002). Self-regulation and self-management in asthma: exploring the role of illness perceptions and treatment beliefs in explaining non-adherence to preventive medication. *Psychology and Health*, 17, 17-32.

Horton-Ausknecht, J. R., Mitzdorf, U., & Melchart, D. (2000). The effect of hypnosis therapy on the symptoms and disease activity in Rheumatoid Arthritis. *Psychology & Health*, 14, 1089-1104.

Houssein A. H. (1998). PhD Thesis, University of London.

Houssein, D. A., Jonsson, T., & Scott, D. L. (1997). IgA rheumatoid factor in rheumatoid arthritis. *Journal of Rheumatology*, 24, 2119-2122.

Huskisson, E. (1974). Measurement of Pain. *Lancet*, 2, 1127-1131.

Huskisson, E. (1983). Visual analogue scales. In R. Melzack (Ed.), *Pain Measurement and Assessment*. New York: Raven Press.

Huyser, B. A., & Parker, J. C. (1998). Stress and rheumatoid arthritis: an integrative review. *Arthritis Care and Research*, 11, 135-145

Huyser, B. A., & Parker, J. C., (2002). Rheumatic diseases. In T. J. Boll & S. B. Johnson, (Eds), *Handbook of clinical health psychology: Volume 1. Medical disorders and behavioral application* (pp. 399-441). Washington, DC, US: American Psychological Association.

Huyser, B. A., Parker, J. C., Thoreson, R., Smarr, K. L., Johnson, J. C., & Hoffman, R. (1998). Predictors of subjective fatigue among individuals with rheumatoid arthritis. *Arthritis and Rheumatism*, 41, 2230-2237.

Ivashkiv, L. B. (1996). Cytokine expression and cell activation in inflammatory arthritis. *Advanced Immunology*, 63, 337-376.

Jantti, J., Aho, K., Kaarela, K., & Kautiainen, H. (1999). Work disability in an inception cohort of patients with seropositive rheumatoid arthritis: a 20 year study. *Rheumatology*, 38, 1138-1141.

Jenkinson, C., Stradling, J., & Peterson, S. (1998). How should we evaluate health status? A comparison of three methods in patients presenting with obstructive sleep apnoea. *Quality of Life Research*, 7, 95-100.

Jensen, Mark P. (2002). Enhancing motivation to change in pain treatment. D. C. Turk, R. J. Gatchel (eds). *Psychological approaches to pain management: A practitioner's handbook* (2nd ed., pp. 71-93). New York: Guilford Press

Jensen, M. P., & Karoly, P. (1992) Self-report scales and procedures for assessing pain in adults. In D. C. Turk & R. Melzack (Eds). *Handbook of Pain Assessment* (pp. 135-131). New York: The Guilford Press.

- Jenson, M. P., Turner, J. A., Romano, J. M., & Karoly, P. (1991). Coping with chronic pain: a critical review of the literature. *Pain*, 47, 249-283.
- Jerusalem, M., & Schwarzer, R. (1992). Self- efficacy as a resource factor in stress appraisal processes. In R. Schwarzer (Ed.), *Self- efficacy: Thought control of action* (pp. 195– 213). Washington: Hemisphere.
- Jivoff L. (1975) Rehabilitation and rheumatoid arthritis. *Bull. Rheum. Dis.*, 26, 838-841.
- Johnston, M. (1996). Models of disability. *The Psychologist*, 9, 205-210.
- Kaarela, K. (1985). Prognostic factors and diagnostic criteria in early rheumatoid arthritis. *Scandinavian Journal of Rheumatology*, 57, 1–54.
- Kaplan, S., & Kozin, F. (1981). A controlled study of group counseling in rheumatoid arthritis. *Journal of Rheumatology*, 8, 91-99.
- Kaptein, A. A., Scharloo, M., Helder, D. I., Kleijn, W.C., Van Korlaar, I. M. & Woertman, M. (2003). Representations of chronic illness. In L.D. Cameron, & H. Leventhal (Eds.). *The self-regulation of health and illness behaviours*. New York: Routledge.
- Katz, P. P. (1995). The impact of rheumatoid arthritis on life activities. *Arthritis Care & Research*, 8, 272-278.
- Katz, P. P. (1998). The stresses of rheumatoid arthritis: Appraisals of perceived impact and coping efficacy. *Arthritis Care and Research*, 11, 9-22.
- Katz, P. P., & Yelin, E. H. (1993). Prevalence and correlates of depressive symptoms among persons with RA. *Journal of Rheumatology*, 20, 790-796.
- Katz, P. P., & Yelin, E. H. (1995). The development of depressive symptoms among women with rheumatoid arthritis: The role of function. *Arthritis & Rheumatism*, 38, 49-56.
- Kazis, L. E., Anderson, J. J., & Meenan, R. F. (1989). Effect sizes for interpreting changes in health status. *Medical Care*, 27, 178-189.
- Keefe, F. J., Affleck, G., Lefebvre, J. C., Starr, K., Caldwell, D. S., & Tennen, H (1997a) Pain coping strategies and coping efficacy in rheumatoid arthritis: A daily process analysis. *Pain*, 69, 35-42.
- Keefe, F. J., & Bonk, V. (1999). Psychosocial assessment of pain in patients having rheumatic diseases. *Rheumatic Disease Clinics of North America*, 25, 81- 103.
- Keefe, F. J., Brown, G. K., Wallston, K. A., & Caldwell, D. S. (1989). Coping with rheumatoid arthritis pain: catastrophising as a maladaptive strategy. *Pain*, 37, 51-56.

- Keefe, F. J., Caldwell, D. S., Baucom, D., Salley, A., Robinson, E., Timmons, K., Beupre, P., Weisberg, J., & Helms, M. (1996). Spouse-assisted coping skills training in the management of osteoarthritic knee pain. *Arthritis Care & Research*, 9, 279-291.
- Keefe, F. J., Caldwell, D. S., Martinez, S., Nunley, J., Beckham, J., & Williams, D.A. (1991). Analysing pain in rheumatoid arthritis patients: Pain coping strategies in patients who have had knee replacement surgery. *Pain*, 46, 153-160.
- Keefe, F. J., Caldwell, D. S., Queen, K. T., Gil, K. M., Martinez, S., Crisson, J. E., Ogden, W., & Nunley, J. (1987). Pain coping strategies in osteoarthritis patients. *Journal of Consulting and Clinical Psychology*, 55, 208-212.
- Keefe, F. J., Caldwell, D. S., Williams, D. A., Gil, K. M., et al., (1990a). Pain Coping skills training in the management of osteoarthritic knee pain: a comparative study. *Behavior Therapy*, 21, 49-62.
- Keefe, F. J., Caldwell, D. S., Williams, D. A., Gil, K. M. et al., (1990b). Pain Coping skills training in the management of osteoarthritic knee pain-II: follow-up results. *Behavior Therapy*, 21, 435-447.
- Keefe, F. J., Kashikar-Zuck, S., Robinson, E., Salley, A., Beupre, P., Caldwell, D., Baucom D., & Haythornthwaite J. (1997b). Pain coping strategies that predict patients' and spouses' ratings of patients' self-efficacy. *Pain*, 73, 191-199.
- Keefe, F. J., Salley, A. N., & Lefebvre, J. C. (1992). Coping with pain: conceptual concerns and future directions, *Pain*, 51, 131-134.
- Keefe, F. J., Smith, S. J., Buffington, A. L., Gibson, J., Studts, J. L., & Caldwell, D. S. (2002). Recent advances and future directions in the biopsychosocial assessment and treatment of arthritis. *Journal of Consulting & Clinical Psychology*, 70, 640-55.
- Keefe, F. J., & Van Horn. Y. (1993). Cognitive-behavioural treatment of RA pain. *Arthritis Care & Research*, 6, 213-232.
- Kelley, J. E., Lumley, M. A., & Leisen, J. C. (1997). Health effects of emotional disclosure in rheumatoid arthritis patients. *Health Psychology*, 16, 331-40.
- Kelsey, J. L. (1982) *Epidemiological aspects of musculoskeletal disorders. Monographs in Epidemiology and biostatistics, Vol. 3*. New York: OUP
- Kemp, S. , Morley, S., & Anderson, E. (1999) . Coping with epilepsy: do illness representations play a role? *British Journal of Clinical Psychology* , 38 , 43-58.
- Kirwan, J. R. (1997). The relationship between synovitis and erosions in rheumatoid arthritis. *Rheumatology*, 36, 225-228.
- Kirwan J. R. (2001). Links between radiological change, disability and pathology in rheumatoid arthritis. *Rheumatology* 28, 881-6.

- Kirwan, J. R., & Reeback, J. S. (1986). Stanford Health Assessment Questionnaire modified to assess disability in British patients with RA. *British Journal of Rheumatology*, 25, 206-209.
- Kludt, C. J. (2000). Effects of disease activity, neuroticism, and minor stress on pain perception in two immunologically distinct subgroups of patients with rheumatoid arthritis. Unpublished PhD thesis, The Chicago Medical School, US
- Kraaimaat, F. W., Brons, M. R., Geenen, R., & Bijlsma, J. W. J. (1995c). The effect of cognitive behavior therapy in patients with rheumatoid arthritis. *Behaviour Research & Therapy*, 33, 487-495.
- Kraaimaat, F.W., Van Dam-Baggen, R. M. J., & Bijlsma, J. W. J. (1995a). Depression, anxiety and social support in rheumatoid arthritic women without and with a spouse. *Psychology and Health*, 10, 387-396
- Kraaimaat, F. W., Van Dam-Baggen, R. M. J., & Bijlsma, J. W. J. (1995b). Association of social support and the spouse's reaction with psychological distress in male and female patients with rheumatoid arthritis. *Journal of Rheumatology*, 22, 644-648.
- Kraan, M. C., Versendaal, H., Jonker, M., Bresnihan B, Post, W. J., T-Hart, B. A., Breedveld, F. C., & Tak, P. P. (1998). Asymptomatic synovitis precedes clinically manifest arthritis. *Arthritis Rheum.*, 41, 1481-1488.
- Kraemer, H. C., & Thiemann, S., (1987). How many subjects? *Statistical power analysis in research*. 119.
- Kvien, T. K. (2002) Epidemiology of disability in rheumatoid arthritis. *Rheumatology*. 41, 121-3.
- Kvien, T. K., Glennås, A, Knudsrød, O. G., Smedstad, L. M., Mowinckel, P., & Førre, Ø. (1997). The prevalence and severity of rheumatoid arthritis in Oslo. Results from a county register and a population survey. *Scandinavian Journal of Rheumatology*, 26, 412-8.
- Labovitz, S. (1971). In defense of assigning numbers to ranks. *American Sociological Review*, 36, 521-522.
- Lanza, A. F., & Revenson, R. A. (1993). Social support interventions for rheumatoid arthritis patients: the cart before the horse? *Health Education Quarterly*, 20, 97-117.
- Lavigne, J. V., Ross, C. K., Berry, S. L., Hayford, J. R., & Pachman, L. M. (1992). Evaluation of a psychological treatment package for treating pain in juvenile rheumatoid arthritis. *Arthritis Care and Research*, 5, 101-110.
- Lawrence, J. S. (1977). *Rheumatism in populations*. London: Heinemann.
- Lazarus, R. S. (1993). Coping theory and research: past, present and future. *Psychosom Med*, 55, 234-47.

- LeDoux, J. E. (1987). Emotion. In F. Plum (Ed.). *Handbook of Physiology, Section 1. (Higher Functions of the Brain, vol 5)*. Bethesda: American Psychological Association.
- Leventhal, H., Brissette, I., & Leventhal, E. A. (2003). The common-sense model of self-regulation of health and illness. In L.D. Cameron, & H. Leventhal (eds.). *The self-regulation of health and illness behaviours*. New York: Routledge
- Leventhal, H., & Cameron, L. (1987). Behavioural theories and the problem of compliance. *Patient Education and Counselling*, 10, 117-138.
- Leventhal, H., Diefenbach, M., & Leventhal, E.A. (1992a). Illness cognition: using common sense to understand treatment adherence and affect cognition interactions. *Cognitive Therapy & Research*, 16, 143-163.
- Leventhal, H., Leventhal, A. E., & Schaefer, P. M. (1992b). Vigilant coping and health behaviour. In M. G. Ory, R. P. Abeles & P. D. Lipman, (Eds.), *Ageing, health and behaviour*, (pp. 109-140). Newbury Park: Sage.
- Leventhal, H., Meyer, D. & Nerenz, D. (1980). The commonsense model of illness danger. In S. Rachman (Ed.). *Contributions to Medical Psychology, Vol. 2*. (pp. 7-30). New York: Pergamon Press.
- Leventhal, H., Nerenz, D. R., & Steel, D. J. (1984). Illness representations and coping with health threats. In Baum and Taylor (Eds.) *Handbook of Psychology and Health*, (pp. 219-252). Hillsdale, NJ: Lawrence Elbaum Associates.
- Liebing, E., Pfingsten, M., Bartmann, U., Rueger, U., & Schuessler, G. (1999). Cognitive-behavioral treatment in unselected rheumatoid arthritis outpatients. *Clinical Journal of Pain*, 15, 58-66.
- Lindroth, Y., Bauman, A., Barnes, C., McCredie, M., & Brooks, P. (1989). A controlled evaluation of arthritis education. *British Journal of Rheumatology*, 28, 7-12.
- Lindroth, Y., Brattstroem, M., Bellman, I., Ekestaf, G., Olofsson, Y., Stroembeck, B., Stenshed, B., Wikstroem, I., Nilsson, J. A., & Wolheim, F. A. (1997). A problem-based education program for patients with rheumatoid arthritis: Evaluation after three and twelve months. *Arthritis Care & Research*, 10, 325-332.
- Linos, A., Worthington, J. W., O'Fallon, W. M., & Kurland, L. T. (1980). The epidemiology of rheumatoid arthritis in Rochester, Minnesota: a study of incidence, prevalence and mortality. *American Journal of Epidemiology*, 111, 97-98.
- Llabre, M. M., Spitzer, S. B., Saab, P. G., Ironson, G. H., & Schnerderman, N. (1991). The reliability and specificity of delta versus residualized change as measure of cardiovascular reactivity to behavioural changes. *Psychophysiology*, 28, 701-711.
- Lorig, K. (1995). Patient Education: treatment or nice extra. *British Journal of Rheumatology*, 34, 703-706.

- Lorig, K. R., Chastain, R. L., Ung, E., Shoor, S., & Holman, H. R. (1989a). Development and evaluation of a scale to measure perceived self efficacy in people with arthritis. *Arthritis & Rheumatism*, 32, 37-44.
- Lorig, K.R., Cox, T., Cuevas, Y., Kraines, R.G., & Britton, M.C. (1984). Converging and diverging beliefs about arthritis: Caucasian patients, Spanish speaking patients and physicians. *The Journal of Rheumatology*, 11, 76-79.
- Lorig, K., Feigenbaum, P., Regan, C., Ung, E., Chastain, R. L., & Holman, H. R. (1986). A comparison of lay-taught and professional-taught arthritis self-management courses. *Journal of Rheumatology*, 13, 763-767.
- Lorig, K. & Fries, J. F. (1990). *The Arthritis helpbook*. Addison-Wesley Pub Co.
- Lorig, K. & Gonzalez, V. (1992). The integration of theory with practice: a 12 year case study. *Health Education Quarterly*, 19, 355-368.
- Lorig, K., & Holman, H. R. (1989). Long-term outcomes of an arthritis self-management study. Effects of reinforcement efforts. *Social Science & Medicine*, 29, 221-224.
- Lorig, K., & Holman, H. R. (1993). Arthritis Self-Management Studies: A Twelve-Year Review. *Health Education Quarterly*, 20, 17-28
- Lorig, K., Konkol, L., & Gonzalez, V. (1987). Arthritis Patient Education: A Review of the Literature. *Patient Education and Counselling*, 10, 207-252.
- Lorig, K., Lubeck, D., Kraines, R., Seleznick, M., & Holman, H. R. (1985). Outcomes of self-help education for patients with arthritis. *Arthritis & Rheumatism*, 28, 680-685.
- Lorig, K., Mazonson, P., & Holman, H. (1993). Evidence suggesting that health education for self-management in patients with chronic arthritis has sustained health benefits while reducing care costs. *Arthritis & Rheumatism*, 36, 439-446.
- Lorig, K., Seleznick, M., Lubeck, D., Ung, E., Chastain, R. L., & Holman, H. R. (1989b). The beneficial outcomes of the arthritis self-management course are not adequately explained by behaviour change. *Arthritis & Rheumatism*, 32, 91-95.
- Lorig, K. R., Sobel, D. S., Stewart, A. L., Brown, B. W. Jr., Bandura, A., Ritter, P., Gonzalez, V. M., Laurent, D. D., & Holman, H. R. (1999). Evidence suggesting that a chronic disease self-management program can improve health status while reducing hospitalization. *Medical Care*, 37, 5-14.
- Lubeck, D. P. (1995). The economic impact of arthritis. *Arthritis Care and Research*, 8, 304-310.
- Maers, S., & Gebhardt, W. (2000). Self-regulation and health behavior: the health behavior model. In M. Boekaerts, P.R. Pintrich and M. Zeider (eds). *Handbook of self-regulation* (pp. 343-368). San Diego, CA: Academic Press.

Maisiak, R., Austin, J., & Heck, L. (1996). Health outcomes of two telephone interventions for patients with rheumatoid arthritis or osteoarthritis. *Arthritis & Rheumatism*, 39, 1391-1399.

Maisiak, R., Austin, J. S., West, S. G., & Heck, L. (1996). The effect of person-centered counseling on the psychological status of persons with systemic lupus erythematosus or rheumatoid arthritis: a randomized, controlled trial. *Arthritis Care and Research*, 9, 60-66.

Masi, A. T. (1983). Articular patterns in the early course of rheumatoid arthritis. *American Journal of Medicine*, 75 (6A), 16-26.

Medical Research Council (2000). *A framework for development of RCT's for complex interventions to improve health*. Retrieved June 11th, 2003 from www.mrc.ac.uk/prn/pdf-mrc_cpr.pdf

McCarty, D. (1974). Selected approaches of synovial membrane physiology. *Arthritis & Rheumatism*, 11, 289-96.

McClenahan, R., & Weinman, J. (1998). Determinants of carer distress in non-acute stroke. *International Journal of Language and Communication Disorders*, 33, 138-143.

McCracken, L. M. (1991). Cognitive Behavioural Treatment of Rheumatoid Arthritis: A Preliminary View of Efficacy and Methodology. *Annals of Behavioural Medicine*, 13, 57-65.

McEntegart, A., Morrison, E., Capell, H. A., Duncan, M. R., Porter, D., Madhok, R et al., (1997). Effect of social deprivation on disease severity and outcome in patients with rheumatoid arthritis. *Annals of Rheumatic Diseases*, 56, 410-413.

McFarlane, A. C., & Brooks, P. M. (1988). Determinants of disability in rheumatoid arthritis. *British Journal of Rheumatology*, 27, 7-14.

McFarlane, A. C., & Brooks, P. M. (1990). Psychoimmunology and rheumatoid arthritis: Concepts and methodologies. *International Journal of Psychiatry in Medicine*, 20, 307-322.

McIntosh, E. (1996). The cost of rheumatoid arthritis. *British Journal of Rheumatology*, 35, 781-790.

McNair, D., Lorr, R., & Dropplemen, L. (1992). *Manual for the Profile of Mood States*. San Diego: Education and Industrial Testing Service.

Mahat, G. (1997). Perceived stressors and coping strategies among individuals with rheumatoid arthritis. *Journal of Advanced Nursing*, 25, 1144-1150.

Malik, N., Greenfield, B. W., Wahl, A. F., & Kiener, P. A. (1996). Activation of human monocytes through CD40 induces matrix metalloproteinases. *Journal of Immunology* 156, 3952-3960.

- Manne, S. L., & Zautra, A. J. (1989). Spouse criticism and support: their association with coping and social adjustment among women with RA. *Journal of Personality and Social Psychology*, 56, 608-617.
- Manne, S. L., & Zautra, A. J. (1990). Couples coping with chronic illness: women with rheumatoid arthritis and their healthy husbands. *Journal of Behavioural Medicine*, 13, 327-342.
- Manne, S. L., & Zautra, A. J. (1992). Coping with Arthritis: Current Status and Critique. *Arthritis and Rheumatism*, 35, 1273-1279.
- Martin, J., & White, A. (1988). *Financial circumstances of disabled adults living in private households*. London: HMSO.
- Marks, R. (2001). Efficacy theory and its utility in arthritis rehabilitation: Review and recommendations, *Disability & Rehabilitation*, 23, 271-280.
- Mason, J. H., Anderson, J. J., & Meenan, R. F. (1988). A model of health status for rheumatoid arthritis: a factor analysis of the Arthritis Impact Measurement Scales. *Arthritis and Rheumatism*, 31, 714-720.
- Mau, W., Bornmann, M., Weber, H., Weidemann, H. F., Hecker, H., & Raspe, H. H. (1996). Prediction of permanent work disability in a follow-up study of early rheumatoid arthritis: results of a tree structured analysis using RECPAM. *British Journal of Rheumatology*, 35, 652-9.
- Meenan, R., Anderson, J. J., Kazis, L. E., Egger, M. J., Altz-Smith, M., Samuelson, J. R. C.O., Willkens, R. J., Solsky, M. A., Hayes, S. P., Blocka, K. L., Weinstein, A., Guttadauria, M., Kaplan, S. B., & Klippel, J. (1984). Outcome assessment in clinical trials; evidence for the sensitivity of a health status measure. *Arthritis & Rheumatism*, 27, 1344-1352.
- Meenan, R., Gertman, P., & Mason, J. (1980). Measuring health status in arthritis: The Arthritis Impact Measurement Scale (AIMS). *Arthritis & Rheumatism*, 23, 146-152.
- Meenan, R., Gertman, P., Mason, J., & Dunaif, R. (1982). The arthritis impact measurement scale: further investigation of a health status measure. *Arthritis & Rheumatism*, 25, 1048-1053.
- Meenan, R., Mason, J., Anderson, J., Guccione, A., & Kazis, L. (1992). AIMS2: The content and properties of a revised and expanded arthritis impact measurement scales Health status questionnaire. *Arthritis & Rheumatism*, 35, 1-10.
- Melzack, R. (1975). The McGill Pain Questionnaire- major properties and scoring methods. *Pain*, 1, 277-99
- Melzack, R., & Wall, P. (1996). *The Challenge of Pain*. (2nd Ed.). Pelican books. London: Penguin

- Merskey, H. (1996). Psychological medicine, pain, and musculoskeletal disorders. *Rheumatic Disease Clinics of North America*, 22, 623-637.
- Meyer, D., Leventhal, H. & Gutmann, M. (1985). Common-sense models of illness: the example of hypertension. *Health Psychology*, 4, 115-135.
- Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change* (2nd ed). New York: Guilford Press.
- Mitchell, D. M., Spitz, P. W., Young, D. Y., Bloch, D. A., McShane, D. J., & Fries, J. F. (1986). Survival, prognosis, and causes of death in rheumatoid arthritis. *Arthritis Rheum* 29, 706-14.
- Mitchell, J., Burkhauser, R., & Pincus, T. (1988). The importance of age, education and comorbidity in the substantial earnings losses of individuals with symmetric polyarthritis. *Arthritis and Rheumatism*, 31, 348-357
- Moorey, S., Greer, S., Gorman, C., Rowden, L., Tunmore, R., Robertson, B., & Bliss, J. (1991). The factor structure and factor stability of the Hospital Anxiety and Depression Scale in patients with cancer. *British Journal of Psychiatry*, 158, 225-259.
- Moreno, I., Valenzuela, A., Garcia, A., Yelamos, J., Sanchez, B., & Hernanz, W. (1996). Association of the shared epitope with radiological severity of rheumatoid arthritis. *Journal of Rheumatology*, 23, 6-9.
- Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*, 80, 1-13
- Moss-Morris, R., Petrie, K. J., & Weinman, J. (1996). Functioning in chronic fatigue syndrome: do illness perceptions play a regulatory role? *British Journal of Health Psychology*, 1, 15-25.
- Moss-Morris, R., Weinman, J., Petrie, K. J., Horne, R., Cameron, L. D., & Buick, D. (2002). The Revised Illness Perception Questionnaire (IPQ-R). *Psychology and Health*, 17, 1-16.
- Möttönen, T., Paimela, L., Leirisalo-Repo, M., Kautiainen, H., Ilonen, J., & Hannonen, P. (1998). Only high disease activity and positive rheumatoid factor indicate poor prognosis in patients with early rheumatoid arthritis treated with 'sawtooth' strategy. *Annals of Rheumatic Disability* 57, 533-539.
- Mullen, P. D., Laville, E. A., Biddle, A. K., & Lorig, K. (1987). Efficacy of psychoeducational interventions on pain, depression, and disability in people with arthritis: a meta-analysis. *Journal of Rheumatology*, 14, 33-39.
- Murphy, H., Dickens, C., Creed, F., & Bernstein, R. (1999). Depression, illness perception and coping in Rheumatoid Arthritis. *Journal of Psychosomatic Research* 46, 155-64.

- Myers, R. E. (2003). Self-regulation and decision-making about cancer screening. In L.D. Cameron, & H. Leventhal (Eds.). *The self-regulation of health and illness behaviours*. New York: Routledge.
- Newman, S. (1998). Psychological aspects of rheumatic disease. In P. J. Maddison, D. A. Isenberg, P. Woo & D. N. Glass (Eds.). *Oxford Textbook of Rheumatology*, (2nd Ed., Vol 1, pp. 63-72). New York: Oxford University Press.
- Newman, S., Fitzpatrick, R., Lamb, R., & Shipley, M. (1990). Patterns of coping in RA. *Psychology and Health*, 4, 187-200.
- Newman, S., & Mulligan, K. (2000). The psychology of rheumatic diseases. *Bailliere's Clinical Rheumatology*, 14, 773-786.
- Newman, S. P., Fitzpatrick, R., Lamb, R., & Shipley, M. (1989). The origins of depressed mood in rheumatoid arthritis. *Journal of Rheumatology*, 16, 740-744.
- Newman, S. P., & Revenson, T.A. (1993). Coping with rheumatoid arthritis. *Bailliere's Clinical Rheumatology*, 7, 259-80.
- Nicassio, P., Radojevic, V., Weisman, M., Culbertson, A., Lewis, C., & Clemmay, P. (1993). The role of helplessness in the response to disease modifying drugs in RA. *Journal of Rheumatology*, 20, 1114-1120.
- Nicassio, P. M., & Wallston, K. A. (1992). Longitudinal relationships among pain, sleep problems, and depression in rheumatoid arthritis. *Journal of Abnormal Psychology*, 101, 514-520.
- Nicassio, P. M., Wallston, K. A., Callahan, L. F., Herbert, M., & Pincus, T. (1985). The measurement of helplessness in rheumatoid arthritis: the development of the Arthritis Helplessness Index. *Journal of Rheumatology*, 12, 462-467.
- Nicholas, M., Wilson, P., & Goyen, J. (1991). Comparison of operant-behavioural and cognitive behavioural group treatment with and without relaxation training for chronic low back pain. *Behaviour, Research and Therapy*, 29, 225-238.
- Northern Ireland Statistics and Research Agency. (2000) Registrar General Northern Ireland Annual Report 2000. Retrieved April 30, 2002, from www.nisra.gov.uk
- Office of Health Economics. (2001). OHE *Health Statistics Compendium 2001*. Retrieved May 22nd, 2002 from www.ohe.org/compendi.htm
- Office of National Statistics. (2000). *Review of the Registrar General on deaths by cause, sex and age, in England and Wales*. Retrieved May 22nd, 2002 from www.statistics.gov.uk
- Office for national statistics (2001). *United Kingdom Health Statistics*. Retrieved October 2nd, 2002, from <http://www.arc.org.uk>.

- O'Leary, A., Shoor, S., Lorig, K., & Holman, H. R. (1988). A cognitive-behavioural treatment for RA. *Health Psychology*, 7, 527-544.
- Ollier, W. E., & MacGregor, A. (1995). Genetic epidemiology of rheumatoid disease. *British Medical Bulletin*, 51, 267-85.
- Orbell, S., Johnston, M., Rowley, D., Espley, A., & Davey, P. (1998). Cognitive representations of illness and functional and affective adjustment following surgery for osteoarthritis. *Social Science and Medicine*, 47, 93-102.
- Osterweis, M., Kleinman, A., & Mechanic, D. (1987). *Disability and pain: Clinical, behavioral, and public policy perspectives*. Washington, DC: National Academy Press.
- Panayi, G. S., Lanchbury, J. S., & Kingsley, G. H. (1992). The importance of the T cell in initiating and maintaining the chronic synovitis of rheumatoid arthritis. *Arthritis Rheum* 35, 729-35.
- Papageorgiou, A. C., & Badley, E. M. (1989). The quality of pain in arthritis: the words used to describe overall pain and pain in individual joints at rest and on movement. *Journal of Rheumatology*, 16, 106.
- Park, D. C. (1994). Self-regulation and control of rheumatic disorders. In S. Maes, H. Leventhal & M. Johnston (eds), *International Review of Health Psychology*, 3. 189-217.
- Parker, J., Frank, R., Beck, N., Finan, M., Walker, S., & Hewett, J. E. (1988a). Pain in rheumatoid arthritis: relationship to demographic, medical, and psychological factors. *Journal of Rheumatology*, 15(3), 433-437.
- Parker, J. C., Frank, R. G., Beck, N. C., Smarr, K. L., Buescher, K. L., & Phillips, L. R. (1988b). Pain management in rheumatoid arthritis patients: a cognitive-behavioral approach. *Arthritis and Rheumatism*, 31, 593-601.
- Parker, J. C., Iverson, G. L., Smarr, K. L., & Stucky-Ropp, R. C. (1993). Cognitive-behavioural approaches to pain management in rheumatoid arthritis. *Arthritis Care & Research*, 6, 207-212.
- Parker, J. C., Singsen, B. H., Hewett, J. E., Walker, S. E., Hazelwood, S. E., Hall, P. J., Holsten, D. J., & Rodon, C. M. (1984). Educating patients with rheumatoid arthritis: a prospective analysis. *Archives of Physical Medicine Rehabilitation*, 65, 771-774.
- Parker, J., Smarr K., Anderson S., Hewett, J., Walker, S., & Bridges, A. (1992). Relationship of changes in helplessness and depression to disease activity in rheumatoid arthritis. *Journal of Rheumatology*, 19, 1901-1905.
- Parker, J. C., Smarr, K. L., Angelone, E. O., Mothershead, P. K., Lee, B. S., Walker, S. E., Bridges, A. J., & Caldwell, C. W. (1992). Psychological factors, immunologic

- activation, and disease activity in rheumatoid arthritis. *Arthritis Care and Research*, 5, 196-201.
- Parker, J. C., Smarr, K. L., Buckelew, S. P., Stucky-Ropp, R. C., Hewett, J. E., Johnson, J. C., et al. (1995). Effects of stress management on clinical outcomes in rheumatoid arthritis. *Arthritis Rheum*; 38, 1807-18.
- Parker, J. C., Smarr, K. L., Buescher, K. L., Phillips, L. R., Frank, R. G., Beck, N. C., Anderson, S. K. & Walker, S. E. (1989). Pain Control and Rational Thinking - implications for rheumatoid arthritis. *Arthritis & Rheumatism*, 32, 984-990.
- Parker, J. C., Smarr, K. L., Walker S. E., Hagglund, K. J., Anderson, S. K., & Hewett, J. E. (1991). Biopsychosocial parameters of disease activity in rheumatoid arthritis: a prospective study of 400 patients. *Arthritis Care and Research*, 4, 73-80.
- Parker J. C., & Wright G. E. (1995). The implications of depression for pain and disability in rheumatoid arthritis. *Arthritis Care & Research*, 8, 279-283.
- Peck, J., Smith, T., Ward, J., & Milano, R. (1989). Disability and depression in RA: a multi-trait, multi-method investigation. *Arthritis & Rheumatism*, 32, 1100-1106.
- Penninx, B. W. J. H., Tilburg, T. V., Deeg, D. J. H., Kriegsman, D. M. W., Boeke, A. J. P., & Van Eijk, J. T. M. (1997). Direct and buffer effects of social support and personal coping resources in individuals with arthritis. *Social Science & Medicine*, 44, 393- 402.
- Petrie, K. J., Broadbent, E., & Meechan, G. (2003). Self-regulatory interventions for improving the management of chronic illness. In L.D. Cameron, & H. Leventhal (Eds.). *The self-regulation of health and illness behaviours*. New York: Routledge.
- Petrie, K. J., Cameron, L. D., Ellis, C. J., Buick, D. L., & Weinman, J. (2002). Changing illness perceptions following myocardial infarction: an early intervention randomised controlled trial. *Psychological Medicine*, 64, 580-586.
- Petrie, K., Moss-Morris, R., & Weinman, J. (1995). The impact of catastrophic beliefs on functioning in chronic fatigue syndrome. *Journal of psychosomatic research*, 39, 31-37.
- Petrie, K. J., Weinman, J., Sharpe, N., & Buckley, J. (1996). Role of patients view of their illness in predicting return to work and functioning after myocardial infarction: A longitudinal study. *British Medical Journal*, 312, 1191-1194.
- Petrie, K. J., & Weinman, J. A. (1997). *Perceptions of health and illness*. Amsterdam: Harwood
- Pimm, T. J. (1989). An exploratory study of the role of perceived control in the emotional impact of chronic disease. Unpublished Diploma in Clinical psychology Dissertation, British Psychological Society.

- Pimm, T. J., Amos, M., & Byron, M. A. (1992). Evaluation of outpatient education programme for people with rheumatoid arthritis. *British Journal of Rheumatology*, 31 (Abstract Supple.), 77.
- Pimm, T. J., Byron, M. A., Curson, D., Hill, S., Hall, J., & Avers, H. (1997). Quality of life in rheumatoid arthritis: an individualized approach. *British Journal of Rheumatology*, 36, (Abstract Supple. 1), 148.
- Pimm, T.J., & Weinman, J. (1998). Applying Leventhal's self-regulation model to adaptation and intervention in rheumatic disease. *Clinical Psychology and Psychotherapy*, 5, 62-75.
- Pincus, T., & Callahan, L. F. (1985). Formal education as a marker for increased mortality and morbidity in rheumatoid arthritis. *Journal of Chronic Diseases*, 38, 973-984.
- Pincus, T. & Callahan, L. F. (1986). Taking mortality in rheumatoid arthritis seriously – predictive markers, socio-economic status and comorbidity. (Editorial.) *Journal of Rheumatology*, 13, 841-5.
- Pincus, T., & Callahan, L. F. (1989). Reassessment of twelve traditional paradigms concerning the diagnosis, prevalence, morbidity and mortality of rheumatoid arthritis. *Scandinavian Journal of Rheumatology*, (Suppl. 79), 67-95.
- Pincus, T., & Callahan, L. F. (1993a). Side effects of Rheumatoid Arthritis: joint destruction, disability and early mortality. *British Journal of Rheumatology*, 32, 28-37.
- Pincus, T., Callahan, L. F. (1993b). What is the natural history of rheumatoid arthritis? *Rheumatic Diseases Clinics of North America*. 19, 123-51.
- Pincus, T., Callahan, L. F., Sale, W.G., Brooks, A. L., Payne, L. E., & Vaughn, W. K. (1984). Severe functional declines, work disability and increased mortality in seventy-five rheumatoid arthritis patients studied over nine years. *Arthritis & Rheumatism*, 27, 864-72.
- Pincus, T., Griffith, J., Pearce, S., & Isenberg, D. (1996). Prevalence of self-reported depression in patients with rheumatoid arthritis. *British Journal of Rheumatology*, 35, 879-883.
- Pincus, T., Marcum, S. B., & Callahan, L. F. (1992). Longterm drug therapy for rheumatoid arthritis in seven rheumatology private practices: II. Second line drugs and prednisone. *Journal of Rheumatology* 19;
- Pincus, T., Swearingen, C., & Wolfe, F. (1999). Toward a Multidimensional Health Assessment Questionnaire (MDHAQ) Assessment of advanced activities of daily living and psychological status in the patient-friendly health assessment questionnaire format. *Arthritis & Rheumatism*, 42, 2220-2230.

- Piper, B. F. (1989). Fatigue: current bases for practice. In S. Funk, E. Tornquist, M. Champagne, L. Copp, & R. Wiese (Eds.), *Key aspects of comfort*. (pp. 187-198). New York: Springer.
- Plant, M. J., Saklatvala, J., Borg, A. A., Jones, P. W., & Dawes, P. T. (1994). Measurement and prediction of radiological progression in early rheumatoid arthritis. *Journal of Rheumatology*, 21, 1808-13.
- Pocock, S. J. (1983). *Clinical Trials: A Practical Approach*. Chichester: Wiley
- Porter, D. R., McInnes, I., Hunter, J., & Capell, H. A. (1994). Outcome of second line therapy in rheumatoid arthritis. *Annals of the Rheumatic Diseases*, 53, 812-5.
- Radojevic, V., Nicassio, P. & Weisman, M. (1992). Behavioural intervention with & without family support for RA. *Behaviour Therapy*, 23, 13-30.
- Rasker, J. J., & Cosh, J. A. (1981). Cause and age at death in a prospective study of 100 patients with rheumatoid arthritis. *Annals of the Rheumatic Diseases*, 40, 115-20.
- Rasker, J. J., & Cosh, J. A. (1984). The natural history of rheumatoid arthritis: a fifteen year follow up study. *Clinical Rheumatology*, 3, 11-20.
- Rasker, J. J., & Cosh, J. A. (1989). Course and prognosis of early rheumatoid arthritis. *Scandinavian Journal of Rheumatology* (Supplement), 79, 45-56.
- Rau, R., & Herborn, G. (1996). Healing phenomena of erosive change in rheumatoid arthritis patients undergoing disease-modifying antirheumatic drug therapy. *Arthritis Rheumatology*, 39, 162-8.
- Ravinder, N., & Feldmann, M. (1998). Immunopathogenesis of rheumatoid arthritis In P. U. J. Maddison, D. A. Isenberg, P. Woo, D. N. Glass, (Eds) . *Oxford Textbook of Rheumatology* (2nd Ed., Vol 1, pp 983-1003). New York: Oxford University Press.
- Reisine, S., Fifield, J., & Winkelman, D. K. (1998). Employment patterns and their effect on health outcomes among women with rheumatoid arthritis followed for 7 years. *Journal of Rheumatology*, 25, 1908-16.
- Reisine, S. T., Goodenow, C., & Grady, K. E. (1987). The impact of rheumatoid arthritis on the homemaker. *Social Science and Medicine*, 25, 89-95.
- Revill, S., Robinson, J., Rosen, M. et al., (1976) The reliability of a linear analogue for evaluating pain. *Anaesthesia* 31, 1191-8
- Revenson, T. A. (1993). The role of social support with rheumatic disease. *Baillieres Clinical Rheumatology*, 7, 377-396.
- Revenson, T. A., & Felton, B. J. (1989). Disability and coping as predictors of psychological adjustment to rheumatoid arthritis. *Journal of Consulting and Clinical Psychology*, 57, 344-348.

- Rhee, S. H., Parker, J. C., Smarr, K. L., Petroski, G. F., Johnson, J. C., Hewett, J. E., Wright, G. E., Multon, K. D., & Walker, S. E. (2000) Stress management in rheumatoid arthritis: What is the underlying mechanism? *Arthritis Care & Research*, 13(6), 435-442.
- Rhind, V. M., Bird, H. A., & Wright, V. (1980). Comparison of clinical assessments of disease activity in RA. *Annals of the Rheumatic diseases*, 39, 135-7.
- Riemsma, R. P., Rasker, J. J., Taal, E. et al., (1998). Fatigue in rheumatoid arthritis: the role of self-efficacy and problematic social support, *British Journal of Rheumatology*, 37(10), 1042-1046.
- Ritchie, D. M., Boyle, J. A., McInnes, J. M. et al. (1968) Clinical studies with an articular index for the assessment of joint tenderness in patients with rheumatoid arthritis. *Quart J Med*, 37, 393-406
- Ropes, M. W., Bennett, G. A., Cobb, S., Jacox, R., & Jessar, R. (1958). A revision of diagnostic criteria for rheumatoid arthritis. *Bull Rheum Dis*. 9, 175-6.
- Roscam, S. (1986). *Application of a Health Locus of Control typology approach toward predicting depression and medical adherence in rheumatoid arthritis*. Unpublished doctoral dissertation. Nashville T.N., Vanderbilt University.
- Rosenstiel, A. & Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment', *Pain*, 17, 33-44.
- Ross, N. (1998). Randomised block design is even more powerful. Retrieved June 9th, 2003 from www.bmj.com/cgi/eletters/317/7155/362#612
- Rothman, A. J., Kelly, K. M., Hertel, A. W., & Salovey, P. (2003). Message frames and illness representations: implications for interventions to promote and sustain healthy behavior. In L.D. Cameron, & H. Leventhal (eds.). *The self-regulation of health and illness behaviours*. New York: Routledge
- Ruta, D. A., Garratt, A. M., Leng, M., Russell, I. T., & MacDonald, L. M. (1994). A new approach to the measurement of quality of life. *The Patient-Generated Index. Medical Care*. 32, 1109-26.
- Rutter, C. L., & Rutter, D. R. (2002) . Illness representation, coping and outcome in irritable bowel syndrome. *British Journal of Health Psychology*, 7(4), 377-392.
- Sakalys, J. A. (1997). Illness behaviour in rheumatoid arthritis. *Arthritis Care & Research*, 10, 229-237
- Scharloo, M., Kaptein, A. A., Weinman, J. A., Hazes, J. M. W., Breedveld, F. C. & Rooijmans, H. G. M. (1999). Predicting Functional Status in Patients with Rheumatoid Arthritis. *The Journal of Rheumatology* 26, 1686-1693

- Scharloo, M. , Kaptein, A. A. , Weinman, J. , Vermeer, B. J., & Rooijmans, H. G. M. (2000a) . Patients' illness perceptions and coping as predictors of functional status in psoriasis: a 1- year follow up. *British Journal of Dermatology* , 142 , 899-907.
- Scharloo, M. , Kaptein, A. A. , Weinman, J. , Willems, L. N. A., & Rooijmans, H. G. M. (2000b) . Physical and psychological correlates of functioning in patients with chronic obstructive pulmonary disease. *Journal of Asthma* , 37 , 17-29.
- Scharloo, M., Kaptein, A. A., Weinman, J., Hazes, J. M., Willems, L. N., Bergman, W., & Rooijmans, B. J. (1998). Illness perceptions, coping and functioning in patients with rheumatoid arthritis, chronic obstructive pulmonary disease and psoriasis. *Journal of Psychosomatic Research*, 44, 573- 585.
- Scheier M. F. & Carver C. S. (2003). Goals and confidence as self-regulatory elements underlying health and illness behavior. In L.D. Cameron, & H. Leventhal (Eds.). *The self-regulation of health and illness behaviours*. New York: Routledge
- Schiaffino, K. M., & Cau, C. D. (1995). Assessing chronic illness representations: The implicit models of illness questionnaire. *Journal of Behavioural Medicine*, 18, 531-548.
- Schiaffino, K. M., & Revenson, T. A. (1992). The role of perceived self-efficacy, perceived control and causal attributions in adaptation to rheumatoid arthritis: distinguishing mediator from moderator effects. *Personality and Social Psychology Bulletin*, 18, 709-718.
- Schiaffino, K. M., Revenson, T. A., & Gibofsky, A.(1991). Assessing the impact of self-efficacy beliefs on adaptation to RA. *Arthritis Care & Research*, 4, 150-157.
- Schiaffino, K. M. , Shawaryn, M. A., & Blum, D. (1998) . Examining the impact of illness representations on psychological adjustment to chronic illness. *Health Psychology*, 17, 262 268.
- Schoenfeld-Smith, K., Petrosici, S. F., Hewett, J. E., Johnson, J. C., Wright, G. E., Smarr, K. L., Walker, S. E., & Parker, J. C. (1996). A biopsychosocial model of disability in rheumatoid arthritis. *Arthritis Care and Research*, 9, 368-375.
- Scholten, C., Brodowicz, T., Graninger, W., Gardavsky, I., Pils, K., Pesau, B., et al. (1999). Persistent functional and social benefit 5 years after a multidisciplinary arthritis training program. *Arch Phys Med Rehabil*, 80, 1282-7.
- Schussler, G. (1992). Coping Strategies and Individual Meanings of Illness. *Social Science and Medicine* 34, 427-432.
- Schwartz, R. (ed) (1992). *Self-efficacy: Thought control of action*. Washington: Hemisphere.
- Scott, D. L., Pugner, K., Kaarela, K., Doyle, D.V., Woof, A., Holmes, J., & Hieke, K. (2000). Review: The links between joint damage and disability in rheumatoid arthritis. *Rheumatology* 39, 122-132.

- Scott, D. L., Smolen, J. S., Kalden, J. R., Van de Putte, L. B., Larsen, A., Kvien, T. K., Schattenkirchner, M., Nash, P., Oed, C., & Loew-Friedrich, I. (2001). European Leflunomide Study Group. Treatment of active rheumatoid arthritis with leflunomide: two year follow up of a double blind, placebo controlled trial versus sulfasalazine. *Annals of the Rheumatic Diseases*, 60, 913-23.
- Scott, D. L., Symmons, D. P. M., Coulton, B. L., & Popert, A. J. (1987). The long-term outcome of treating rheumatoid arthritis: results after 20 years. *Lancet*, 1108-11.
- Seligman, M. E. P (1975). *Helplessness: On depression, development and death*. San Francisco: Freeman.
- Semle, E. L. (1995). Rheumatoid arthritis: new approaches for its evaluation and management. *Arch Phys Med Rehabil*, 76, 190-201.
- Sharpe, A. L (1999). *A blind, randomised, controlled trial of a cognitive and behavioural intervention for recent-onset Rheumatoid Arthritis: Preventing physical and psychological morbidity*. Unpublished PhD thesis, Royal Holloway, University of London.
- Sharpe, L., Sensky, T., & Allard, S. (2001a). The Course of depression in recent onset Rheumatoid Arthritis: The predictive role of disability, illness perceptions, pain and coping. *Journal of psychosomatic research*, 51 (6), 713-719.
- Sharpe L., Sensky, T., Timberlake, N., Ryan B., & Allard S. (2001c). Long-term efficacy of a cognitive behavioural treatment from a randomised controlled trial for patients recently diagnosed with rheumatoid arthritis. *Rheumatology*
- Sharpe, L., Sensky, T., Timberlake, N., Ryan, B., Brewin, C. R., & Allard, S. (2001b). A blind, randomized, controlled trial of cognitive-behavioural intervention for patients with recent onset rheumatoid arthritis: preventing psychological and physical morbidity. *Pain*, 89, 275-83.
- Shearn, M. A., & Fireman, B. H.(1985). Stress management and mutual support groups in rheumatoid arthritis. *The American Journal of Medicine*, 78, 771-775.
- Shifren, K., Park, D. C., Bennett, J. M., & Morrell, R. W. (1999). Do cognitive processes predict mental health in individuals with rheumatoid arthritis? *Journal of Behavioural Medicine*. 22, 529-547.
- Shipley, M., & Newman, S.P. (1993) Psychological aspects of rheumatic diseases. *Bailliere's Clinical Rheumatology*, 7, 215-9.
- Silman, A., Davies, P., Currey, H. L., & Evans, S. J. (1983). Is rheumatoid arthritis becoming less severe? *Journal of Chronic Diseases*, 36, 891-7.
- Silman, A. J., & Hochberg, M. C. (2001). *Epidemiology of the Rheumatic Diseases*. 2nd Edition. Oxford: Oxford Medical Publications.

- Simon, L., Lipman, A. G., Allaire, S. G., Caudill- Slosberg, M., Gill, L. H., Keefe, F. J., et al. (2002) *Guideline for the management of acute and chronic pain in osteo and rheumatoid arthritis*. Glenview, IL: American Pain Society.
- Simon, L. S., & Yocum, D. (2000). New and future drug therapies for rheumatoid arthritis. *Rheumatology*, 39 (suppl. 1), 36-42
- Skevington, S. M. (1986). Psychological aspects of pain in rheumatoid arthritis: A review. *Social Science & Medicine*, 23, 567-575.
- Skevington, S. M. (1993) Depression and causal attributions in the early stages of a chronic painful disease: a longitudinal study of early synovitis. *Psychological Health* 8, 51-64.
- Snaith, R. P., & Zigmond, A. S. (1994). *HADS: Hospital Anxiety and Depression Scale*. Windsor: NFER Nelson.
- Smarr, K. L., Parker, J. C., Wright, G. E. et al. (1997). The importance of enhancing self-efficacy in rheumatoid arthritis. *Arthritis Care and Research*, 10 (1), 18-26.
- Smedstad, L. M., Moum, T., Guillemin, F., Kvien, T. K., Finch, M. B., Suurmeijer, T. P. et al. (1996). Correlates of functional disability in early rheumatoid arthritis: a cross-sectional study of 706 patients in four European countries. *Br. J. Rheumatol*, 35, 746-51.
- Smith, C. A., Dobbins, C. J., & Wallston, K. A. (1991). The mediational role of perceived competence in psychological adjustment to rheumatoid arthritis. *Journal of Applied Social Psychology*, 21, 1218-47.
- Smith, C. A., & Wallston, K. A. (1992). Adaptation in patients with chronic rheumatoid arthritis: Application of a general model. *Health Psychology*, 11, 151-162.
- Smith, H. J., Taylor, R., & Mitchell, A. (2000). A comparison of four quality of life instruments in cardiac patients: SF-36, QLI, QLMI, and SEIQoL. *Heart*, 84, 390-394.
- Smith, T., Christensen, A., Peck, J., & Ward, J. (1994). Cognitive distortion, helplessness and depressed mood in RA: a 4 year longitudinal analysis. *Health Psychology*, 13, 213-217.
- Smith, T. W., Peck, J. R., Milano, R. A., & Ward, J. R. (1988). Cognitive distortion in rheumatoid arthritis: Relation to depression and disability. *Journal of Consulting & Clinical Psychology*, 56, 412-416.
- Smith, T. W., Peck, J. R., & Ward, J. R. (1990). Helplessness and depression in rheumatoid arthritis. *Health Psychology*, 9, 377-389.
- Smolen, J. S., Tohidast-Akrad, M., Gal, A. et al., (1996). The role of T-lymphocytes and cytokines in rheumatoid arthritis. *Scandinavian Journal of Rheumatology*, 25, 1-4.

- Smyth, J. M., Stone, A. A., Hurewitz, A., & Kaell, A. (1999). Effects of writing about stressful experiences on symptom reduction in patients with asthma or rheumatoid arthritis: A randomized trial. *Journal of the American Medical Association*, 281, 1304-1309.
- Soderlin, M. K., Nieminen, P., & Hakala, M. (1998). Functional status predicts mortality in a community based rheumatoid arthritis population. *Journal of Rheumatology*, 25, 1895-9.
- Sokka, T., Kautiainen, H., Mottonen, T., & Hannonen, P. (1999). Work disability in rheumatoid arthritis 10 years after the diagnosis. *Journal of Rheumatology*, 26, 1681-5.
- Spector, T. D., & Scott, D. L. (1988) What happens to patients with rheumatoid arthritis? The longterm outcome of treatment. *Clinical Rheumatology*, 7, 315-30
- Steed, L., Newman, S. P., & Hardman, S. M. C. (1999) . An examination of the self-regulation model in a trial fibrillation. *British Journal of Health Psychology*, 4 (4), 337-347.
- Steinbrocker, O., Traeger, C., & Batterman, R. (1949). Therapeutic criteria in rheumatoid arthritis. *Journal of the American Medical Association*, 140. 659 - 62.
- Steiner, G., Tohidast-Akrad, M., Witzmann G et al., (1999). Cytokine production of synovial T cells in rheumatoid arthritis. *Rheumatology*, 3,: 202-13.
- Stone, A., Kessler, R., & Haythornthwaite, J. (1991). Measuring daily events and experiences: Decisions for the researcher. *Journal of Personality*, 59, 575- 608.
- Stone, A., & Neale, J. (1984). New measure of daily coping: Development and preliminary results. *Journal of Personality and Social Psychology*, 46, 892- 906.
- Strahl, C., Kleinknecht, R. A., & Dinnel, D. L. (2000). The role of pain anxiety, coping, and pain self-efficacy in rheumatoid arthritis patient functioning. *Behaviour Research & Therapy*, 38, 863-873.
- Straughair, S., & Fawcitt, S. (1992). *The road towards independence: The experiences of young people with arthritis*. London: Arthritis Care.
- Strauss, G. D., Spiegel, J. S., Daniels, M., Spiegel, T., Landsverk, J., Roy-Byrne, P., Edelstein, C., Ehlhardt, J., Falke, R., & Hindin, L. (1986). Group Therapies for Rheumatoid Arthritis: A Controlled Study of Two Approaches. *Arthritis and Rheumatism*, 86, 1203-1209.
- Suarez-Almazor, M. E., Soslone, C. L., Saunders, L. D., & Russell, A. S. (1994). Outcome in rheumatoid arthritis. A 1985 inception cohort study. *Journal of Rheumatology*, 21, 1438-46.

- Superio-Cabuslay, E., Ward, M. M. & Lorig, K. R. (1996). Patient education interventions in osteoarthritis and rheumatoid arthritis: A meta-analytic comparison with nonsteroidal antiinflammatory drug treatment. *Arthritis Care & Research*, 9, 292-301.
- Sutej, P. G., & Hadler, N. M. (1991). Current principles of rehabilitation for patients with rheumatoid arthritis. *Clin Orthop*, 265, 116-124.
- Swezey, R. L. (1974). Essentials of physical management and rehabilitation in arthritis. *Seminars in Arthritis and Rheumatism*, 3, 349-368.
- Symmons, D. P. M. (1994). Quantifying progress in arthritis. Arthritis Research Campaign . Retrieved October 1st, 2002, from <http://www.arc.org.uk>
- Symmons, D., & Bankhead, C. (1994). *Health Care Needs Assessment for Musculoskeletal Diseases: The First Step - Estimating the Number of Incident and Prevalent Cases*. Manchester: The Arthritis & Rheumatism Council for Research Epidemiology Research Unit.
- Symmons, D. P. M., Barrett, E. M., Bankhead, C. R., Scott, D. G., & Silman, A. J. (1994). The incidence of rheumatoid arthritis in the United Kingdom: results from the Norfolk Arthritis Register. *British Journal of Rheumatology*, 33, 735-9
- Symmons, G., Turner, R., Webb, P., Asten, E., Barrett, M., Lunt, D., Scott, & Silman, A. (2002). The prevalence of rheumatoid arthritis in the United Kingdom: new estimates for a new century. *Rheumatology*, 41, 793-800.
- Taal, E., Rasker, J. J., Seydel, E. R., & Wiegman, O. (1993a). Health status, adherence with health recommendations, self-efficacy and social support in patients with rheumatoid arthritis. *Patient Education and Counselling*, 20, 63-76.
- Taal, E., Rasker, J. J., & Wiegman, O. (1995). Group education for rheumatoid arthritis patients: a review of the literature. *Seminars in Arthritis and Rheumatism*, 26, 805-816.
- Taal, E., Rasker, J. J., Wiegman, O., Brus, H. L. M. & Riemsma, R. P. (1994). Spouse participation in group self-management education for patients with RA. *Scand. J. Rheumatol. Suppl.*, 97, 24.
- Taal, E., Riemsma, R. P., Brus, H. L., Seydel, E. R., Rasker, J. J., & Wiegman, O. (1993b). Group education for patients with rheumatoid arthritis. *Patient Education & Counselling*, 20, 177-187.
- Tack, B. B. (1990). Self-reported fatigue in rheumatoid arthritis: a pilot study. *Arthritis Care and Research*, 3, 154-157.
- Teasdale, J. D., Segal, Z. V; Williams, J. M. G., Ridgeway, V. A., Soulsby, J. M., Lau, M. A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting & Clinical Psychology*, 68(4), 615-623

- Thompson, P. W., & Pegley, F. S. (1991). A comparison of disability measured by the Stanford Health Assessment Questionnaire disability scales (HAQ) in male and female rheumatoid outpatients. *British Journal of Rheumatology*, 30, 298–300.
- Tones, K. (1997). Health education: evidence of effectiveness. *Archives of Disease in Childhood*, 77(3), 189-91.
- Treasure, T., & MacRae, K.D. (1998). Minimisation: The platinum standard for trials? *British Medical Journal*, 317, 362-363.
- Tucker, M., & Kirwan, J. R. (1991). Does patient education of RA have therapeutic potential? *Annals of the Rheumatic Diseases*, 50, 422-428. (Heberden papers).
- Tugwell, P., & Boers, M. (1993). Developing consensus on preliminary core efficacy endpoints for rheumatoid arthritis clinical trials. *Journal of Rheumatology*, 20, 555-6.
- Turk, D., Rudy, T., & Salovey, P. (1986). Implicit models of illness. *Journal of Behavioural Medicine*, 9, 453-474.
- Uhlig, T., Kvien, T. K., Glennås, A., Smedstad, L. M., & Førre, Ø. (1998). The incidence and severity of rheumatoid arthritis. Results from a county register in Oslo, Norway. *Journal of Rheumatology*, 25, 1078-84.
- Uhlig, T., Kvien, T. K., Haavardsholm, E., & Smedstad, L. M. (1998). Examination of agreement between HAQ and MHAQ in patients with rheumatoid arthritis. *Arthritis & Rheumatism*, 41(Suppl.).
- Uhlig, T., Smedstad, L. M., Vaglum, P., Moum, T., Gerard, N., & Kvien, T. K. (2000). The course of rheumatoid arthritis and predictors of psychological, physical and radiographic outcome after 5 years of follow-up. *Rheumatology*, 39, 732-741.
- Van den Ende, C. H. M., Vliet Vlieland, T. P. M., Munneke, M., & Hazes, J. M. W. (1998). *Dynamic exercise therapy for rheumatoid arthritis* (Cochrane Review). In: The Cochrane Library, Issue 4. Oxford: Update Software. Retrieved May 22nd, 2002, from www.nelh.nhs.uk/cochrane
- Van der Heide, A., Jacobs, J. et al (1994). Physical disability and psychological well-being in recent onset RA. *Journal of Rheumatology*, 21(1), 28-32.
- Van Der Heijde, D. M. F. M., Van Leeuwen, M. A., Van Riel P. L. C. M., & Van de Putte L. B. A. (1995) Radiographic progression on radiographs of hands and feet during the first 3 years of rheumatoid arthritis measured according to Sharp's method (van der Heijde modification). *J. Rheumatol*, 22, 1792-6
- Van Deusen, J., & Harlowe, D. (1987). The efficacy of the ROM Dance Program for adults with rheumatoid arthritis. *Am J Occup Ther*, 41, 90-5.

- Van Lankveld, W., Naring, G., Van der Staak C., Van't Pad Bosch, P., & Van de Putte, L. (1993). Stress caused by rheumatoid arthritis: relation among subjective stressors of the disease, disease status, and well-being. *Journal of Behavioural Medicine*, 16, 309-321.
- Van Lankveld, W., Van't Pad Bosch, P., Van de Putte, L., Naring, G., & Van der Steak, C. (1994). Disease-specific stressors in rheumatoid arthritis: coping and well-being. *British Journal of Rheumatology*, 33, 1067-73.
- Van Leeuwen, M. A., Van der Heijde, D. M. F. M., Van Rijswijk, M.H., et al. (1994). Interrelationships of outcome measures and process variables in early rheumatoid arthritis: a comparison of radiologic damage, physical disability, joint counts, and acute phase reactants. *J Rheumatol*, 21, 425-9.
- Van Schaardenburg, D., Hazes, J. M., De Boer, A., Zwinderman, A. H., Meijers, K. A., & Breedveld, F. C. (1993). Outcome of rheumatoid arthritis in relation to age and rheumatoid factor at diagnosis. *Journal of Rheumatology*, 20, 45-52.
- Van Zeben, D., Hazes J. M., Zwinderman A. H., Cats A., Schreuder G. M., D'Amaro J et al. (1991). Association of HLA-DR4 with a more progressive disease course in patients with rheumatoid arthritis. Results of a followup study. *Arthritis Rheum*, 34, 822-30.
- Van Zeben, D., Hazes, J. M., Zwinderman, A. H., Vandenbroucke, J. P., & Breedveld, F. C. (1993). Factors predicting outcome of rheumatoid arthritis: results of a follow-up study. *Journal of Rheumatology*, 20, 1288-96.
- Vandenbroucke, J. P., Hazeroet, H. M., & Cats, A. (1984). Survival and cause of death in rheumatoid arthritis: a 25 year prospective follow-up. *Journal of Rheumatology*, 11, 158-61.
- Vliet Vlieland, T. P., Buitenhuis, N. A., Van Zeben, D., Vandenbroucke, J. P., Breedveld, F. C., & Hazes, J. M. (1994). Sociodemographic factors and the outcome of rheumatoid arthritis in young women. *Annals of the Rheumatic Diseases*, 53, 803-6.
- Vliet Vlieland, T. P., & Hazes, J. M. (1997). Efficacy of multidisciplinary team care programs in rheumatoid arthritis. *Seminars in Arthritis and Rheumatism*, 27, 110-122
- Wagstaff, S., Smith, O. V., & Wood, P. H. N. (1985). Verbal pain descriptors used by patients with arthritis. *Annals of the Rheumatic diseases*, 44, 262-265.
- Wallston, B. S., Wallston, K. A., Kaplan, G. D., & Maides, S. A. (1976). Development and Validation of the Health Locus of Control (HLC) Scale. *Journal of Consulting and Clinical Psychology*, 44, 580-585.
- Wallston, K. A. (1993). Psychological control and its impact in the management of rheumatological disorders. *Balliere's Clinical Rheumatology*, 7, 281-295.

- Wallston, K. A., Wallston, B. S., Smith, S. & Dobbins C. J. (1987). Perceived control and health. *Current Psychological Research and Reviews*, 6, 5-25
- Ward, M. M., & Leigh, J. P. (1993). Marital status and the progression of functional disability in patients with rheumatoid arthritis. *Arthritis and Rheumatism*, 36, 581-588.
- Ward, M. M., Leigh, J. P., & Fries, J. F. (1993). Progression of functional disability in patients with rheumatoid arthritis. Associations with rheumatology subspecialty care. *Arch. Intern. Med.*, 153, 2229-37.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality & Social Psychology*, 54, 1063-70.
- Wegener, S. T. (1996). Sleep Disturbance. In S. T. Wegener, B. L. Belza, & E. P. Gall (Eds.), *Clinical Care in the Rheumatic Diseases* (pp. 121-124). Atlanta: The American College of Rheumatology
- Weinstein, N. D. (1988). The precaution adoption process. *Health Psychology*, 7, 355-386.
- Weibe, D. J., & Korbel, C. (2003). Defensive denial, affect, and the self-regulation of health threats. In L.D. Cameron, & H. Leventhal (Eds.). *The self-regulation of health and illness behaviours*. New York: Routledge.
- Weinman, J., Heijmans, M., & Figueiras, M. J. (2003). Carer perceptions of chronic illness. In L.D. Cameron, & H. Leventhal (eds.). *The self-regulation of health and illness behaviours*. New York: Routledge
- Weinman, J., Petrie, K., Moss-Morris, R., & Horne, R. (1996). The Illness Perception Questionnaire: A new method for assessing the cognitive representation of illness. *Psychology & Health*, 11, 431-445.
- Weinman, J., Petrie, K. J., Sharpe, N., & Walker, S. (2000). Causal attributions in patients and spouses following first-time myocardial infarction and subsequent lifestyle changes. *British Journal of Health Psychology*, 5, 263-273.
- White, C. A. (2001). *Cognitive behaviour therapy for chronic medical problems: a guide to assessment and treatment in practice*. London: John Wiley & Sons, Ltd.
- Wills, T. A. (1987). Downward comparison as a coping mechanism In C.R. Snyder and C. E. Ford (Eds). *Coping with negative life events*, (pp. 243-68). Plenum, New York.
- Wolfe, F. (2000). A reappraisal of HAQ disability in rheumatoid arthritis. *Arthritis Rheum*, 43, 2751-61.
- Wolfe, F. (2001). Which HAQ is best? A comparison of the HAQ, MHAQ and RA-HAQ, a difficult 8 item HAQ (DHAQ), and a rescored 20 item HAQ (HAQ20): analyses in 2,491 rheumatoid arthritis patients following leflunomide initiation. *Journal of Rheumatology*, 28, 982-9

- Wolfe, F., & Hawley, D. J. (1998). The long term outcomes of rheumatoid arthritis: work disability: a prospective 18 year study of 823 patients. *Journal of Rheumatology*, 25: 2108–17.
- Wolfe, F., Hawley, D. J., & Cathey, M. A. (1991). Clinical and Health Status Measures Over Time: Prognosis and Outcome Assessment in Rheumatoid Arthritis. *The Journal of Rheumatology*, 18, 1290-1297.
- Wolfe, F., Hawley, D.J., & Wilson, K. (1996). The prevalence and meaning of fatigue in rheumatic disease. *Journal of Rheumatology*, 23, 1407–17.
- Wolfe, F., Kleinheksel, S. M., Cathey, M. A., Hawley, D. J., Spitz, P. W., & Fries, J. F. (1988). The clinical value of the Stanford Health Assessment Questionnaire Functional Disability Index in patients with rheumatoid arthritis. *Journal of Rheumatology*, 15, 1480–8.
- Wolfe, F., Mitchell, D. M., Sibley, J. T., Fries, J. F., Bloch, D. A., Williams, C. A., et al. (1994). The mortality of rheumatoid arthritis. *Arthritis Rheum*, 37, 481–94.
- Wolfe, F., & Sharp, J. T. (1998). Radiographic outcome of recent-onset rheumatoid arthritis: a 19-year study of radiographic progression. *Arthritis Rheum*, 41, 1571-82.
- Wolfe, F., & Zwillich, S. H. (1998). The long- term outcomes of rheumatoid arthritis: a 23- year prospective, longitudinal study of total joint replacement and its predictors in 1,600 patients with rheumatoid arthritis. *Arthritis Rheum*, 41, 1072– 82.
- Woolf, A. D., Hall, N. D., Goulding, N. J., Kantharia, B., Maymo, J., Evison, G. et al. (1991). Predictors of the long-term outcome of early synovitis: a 5-year follow-up study. *Br. J. Rheumatol*, 30, 251–4.
- Wollheim, F. A. (1998). Rheumatoid arthritis-the clinical picture. In P. J. Maddison, D. A. Isenberg, P. Woo & D. N. Glass (Eds), *Oxford Textbook of Rheumatology* (2nd Ed, Vol 2, 1004-1030), New York: Oxford University Press.
- World Health Organization. (1980). *International Classification of Impairments, Disabilities, and Handicaps*. Geneva: World Health Organization.
- World Health Organization. (2000). ICIDH- 2: *International Classification of Impairments, Activities and Participation*. Retrieved September 14th, 2000, from: www.who.int/icidh
- Wright, G. E., Parker, J. C., Smarr, K. L., Johnson, J. C., Hewett, J. E., & Walker, S. E. (1998). Age, depressive symptoms, and rheumatoid arthritis. *Arthritis & Rheumatism*, 41, 298- 305.
- Wright, G. E., Parker, J. C., Smarr, K. L., Schoenfeld-Smith, K., Buckelew, S. P., Slaughter, J. R., Johnson, J. C., & Hewett, J. E. (1996). Risk factors for depression in RA. *Arthritis Care and Research*, 9, 264-172.

- Yelin, E. (1992). Arthritis: the cumulative impact of a common chronic condition. *Arthritis and Rheumatism*, 35, 489-497.
- Yelin, E., & Callahan, L. (1995). The economic cost and social and psychological impact of musculoskeletal conditions. *Arthritis & Rheumatism*, 38, 1351- 1362.
- Yelin, E., Meenan, R., Nevitt, M., & Epstein, W. (1980). Work disability in rheumatoid arthritis: effects of disease, social, and work factors. *Annals of Internal Medicine*, 93, 551-6.
- Yocum, D. E. (1999). T cells: pathogenic cells and therapeutic targets in rheumatoid arthritis. *Semin Arthritis Rheum* 29, 27-35.
- Young, A., Dixey, J., Cox, N., Davies, P., Devlin, J., Emery, P., Gallivan, S., Gough, A., James, D., Prouse, P., Williams, P., & Winfield, J. (2000). How does functional disability in early rheumatoid arthritis (RA) affect patients and their lives? Results of 5 years of follow-up in 732 patients from the Early RA Study (ERAS) *Rheumatology* 39, 603-611.
- Young, L. D. (1992). Psychological factors in rheumatoid arthritis. *Journal of Consulting and Clinical Psychology*, 60, 619-627.
- Young, L. D., Bradley, L. A., & Turner, R. A. (1995). Decreases in health care resource utilization in patients with rheumatoid arthritis following a cognitive behavioral intervention. *Biofeedback & Self Regulation*, 20, 259-268.
- Zautra, A. J., Burleson, M. H., Smith, C. A., Blalock, S. J., Wallston, K. A., DeVellis, R. F., DeVellis, B. M., & Smith, T. W. (1995). Arthritis and perceptions of quality of life: An examination of positive and negative affect in rheumatoid arthritis patients. *Health Psychology*, 14, 399-408.
- Zautra, A. J., & Manne, S. L. (1992). Coping with rheumatoid arthritis: a review of a decade of research. *Annals of Behavioural Medicine*, 14, 31-39.
- Zelen, M. (1979). A New Design for Randomised Clinical Trials. *The New England Journal of Medicine*, 300, 1242-1245.
- Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, 67, 361-370.